“DOPPLER SONOGRAPHIC EVALUATION OF INTRARENAL ARTERIES IN ACUTE RENAL OBSTRUCTION”

DISSERTATION SUBMITTED TO
THE TAMIL NADU Dr. M.G.R MEDICAL UNIVERSITY,
CHENNAI
IN PARTIAL FULFILLMENT OF THE REGULATIONS FOR THE AWARD OF DEGREE OF

M.D IN RADIODIAGNOSIS.

BY

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MAY 2018

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This is to certify that the dissertation entitled **"DOPPLER SONOGRAPHIC EVALUATION OF INTRARENAL ARTERIES IN ACUTE RENAL OBSTRUCTION"** is the bonafide original work of **Dr.Shamsa Kappumughath Mohamed** in the **Department of Radiodiagnosis, PSG Institute of Medical Sciences and Research**, Coimbatore in partial fulfillment of the regulations for the award of degree of M.D in Radiodiagnosis.

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This is to certify that the Dissertation work entitled **"DOPPLER SONOGRAPHIC EVALUATION OF INTRARENAL ARTERIES IN ACUTE RENAL OBSTRUCTION"** is the bonafide work of **Dr. Shamsa Kappumughath Mohamed** in the **Department of Radiodiagnosis, PSG Institute of Medical Sciences and Research**, Coimbatore in partial fulfillment of the regulations for the award of degree of M.D in Radiodiagnosis.

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DECLARATION

I, **Dr. SHAMSA KAPPUMUGHATH MOHAMED** solemnly declare that the dissertation titled **"DOPPLER SONOGRAPHIC EVALUATION OF INTRARENAL ARTERIES IN ACUTE RENAL OBSTRUCTION"** was done by me at the **Department of Radiodiagnosis, PSG Institute of Medical Sciences and Research**, Coimbatore during the period from December 2015 to September 2017 under the guidance and supervision of **Dr. ELANGO NAGAPPAN,** Professor, Department of Radio Diagnosis, PSG Institute of Medical Sciences and Research, Coimbatore. This dissertation is submitted to the Tamilnadu Dr.M.G.R. Medical University towards the partial fulfillment of the requirement for the award of M.D. Degree in Radiodiagnosis.

I have not submitted this dissertation on any previous occasion to any University for the award of any degree.

Place : Coimbatore **Dr. Shamsa Kappumughath Mohamed**

Date:

ACKNOWLEDGEMENT

Foremost, I would like to express my sincere gratitude to my professor and HOD **Dr. B. Devanand** and my Guide **Dr. Elango Nagappan** for their ever friendly co-operation which was present throughout the preparation of this work. This work would not have been possible without their guidance, support and encouragement.

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Finally my sincere thanks and gratitude to the associate professors, assistant professors, senior residents, staff and office people for their immense support for carrying out and completing this work.

I dedicate this whole dissertation and all years of hard work to my whole Family and my God Almighty.

Dr. Shamsa Kappumughath Mohamed

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To
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Ref: Project No.15/417

Date: December 30, 2015

Dear Dr Shamsa Kappumughath Mohamed,

Institutional Human Ethics Committee, PSG IMS&R reviewed and discussed your application dated 21.12.2015 to conduct the research study entitled *"Doppler sonographic evaluation of intrarenal arteries in acute renal obstruction"* during the IHEC meeting held on 24.12.2015.

The following documents were reviewed and approved:

1. Project Submission form
2. Study protocol (Version 1 dated 21.12.2015)
3. Informed consent forms (Version 1 dated 21.12.2015)
4. Data collection tool (Version 1 dated 21.12.2015)
5. Permission letter from concerned Heads of the Department
6. Current CVs of Principal investigator, Co-investigators
7. Budget

The following members of the Institutional Human Ethics Committee (IHEC) were present at the meeting held on 24.12.2015 at IHEC Secretariat, PSG IMS & R between 10.00 am and 11.00 am:

Sl. No.	Name of the Member of IHEC	Qualification	Area of Expertise	Gender	Affiliation to the Institution Yes/No	Present at the meeting Yes/No
1	Mr. R. Nandakumar	BA., BL	Legal Expert, Chairperson	Male	No	Yes
2	Dr. S. Bhuvaneshwari (Member-Secretary, IHEC)	MD	Clinical Pharmacology	Female	Yes	Yes
3	Dr. S. Shanthakumari	MD	Pathology, Ethicist	Female	Yes	Yes
4	Dr D Vijaya	M Sc., Ph D	Basic Medical Sciences (Biochemistry)	Female	Yes	Yes

The study is approved in its presented form. The decision was arrived at through consensus. Neither PI nor any of proposed study team members were present during the decision making of the IHEC. The IHEC functions in accordance with the ICH-GCP/ICMR/Schedule Y guidelines. The approval is valid until one year from the date of sanction. You may make a written request for renewal / extension of the validity, along with the submission of status report as decided by the IHEC.

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Following points must be noted:

1. IHEC should be informed of the date of initiation of the study
2. Status report of the study should be submitted to the IHEC every 12 months
3. PI and other investigators should co-operate fully with IHEC, who will monitor the trial from time to time
4. At the time of PI's retirement/intention to leave the institute, study responsibility should be transferred to a colleague after obtaining clearance from HOD, Status report, including accounts details should be submitted to IHEC and extramural sponsors
5. In case of any new information or any SAE, which could affect any study, must be informed to IHEC and sponsors. The PI should report SAEs occurred for IHEC approved studies within 7 days of the occurrence of the SAE. If the SAE is 'Death', the IHEC Secretariat will receive the SAE reporting form within 24 hours of the occurrence
6. In the event of any protocol amendments, IHEC must be informed and the amendments should be highlighted in clear terms as follows:
   a. The exact alteration/amendment should be specified and indicated where the amendment occurred in the original project. (Page no. Clause no. etc.)
   b. Alteration in the budgetary status should be clearly indicated and the revised budget form should be submitted
   c. If the amendments require a change in the consent form, the copy of revised Consent Form should be submitted to Ethics Committee for approval
   d. If the amendment demands a re-look at the toxicity or side effects to patients, the same should be documented
   e. If there are any amendments in the trial design, these must be incorporated in the protocol, and other study documents. These revised documents should be submitted for approval of the IHEC and only then can they be implemented
   f. Any deviation-Violation/waiver in the protocol must be informed to the IHEC within the stipulated period for review
7. Final report along with summary of findings and presentations/publications if any on closure of the study should be submitted to IHEC

Kindly note this approval is subject to ratification in the forthcoming full board review meeting of the IHEC.

Thanking You,

Yours Sincerely,

SECRETARY
PSG IMS & R
COIMBATORE-641 004
INSTITUTIONAL HUMAN ETHICS

Dr Sudha Ramalingam
Alternate Member - Secretary
Institutional Human Ethics Committee

PLAGIARISM CERTIFICATE

This is to certify that this dissertation work titled "**DOPPLER SONOGRAPHIC EVALUATION OF INTRARENAL ARTERIES IN ACUTE OBSTRUCTION"** of the candidate **DR. SHAMSA KAPPUMUGHATH MOHAMED** with registration number **20158103** for the award of degree of MD in the branch of Radiodiagnosis. I personally verified the urkund.com website for the purpose of Plagiarism check. I found that the uploaded thesis file contains introduction to conclusion pages and result shows **8 %** of plagiarism in the dissertation.

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LIST OF ABBREVIATIONS

USG	–	ULTRASONOGRAPHY
CT	–	COMPUTED TOMOGRAPHY
IVP	–	INTRAVENOUS PYELOGRAPHY
KUB	–	KIDNEY, URETER & BLADDER
CDUS	–	COLOR DOPPLER ULTRASOUND
RI	–	RESISTIVITY INDEX
PSV	–	PEAK SYSTOLIC VELOCITY
EDV	–	END DIASTOLIC VELOCITY
DDUS	–	DUPLEX DOPPLER ULTRASOUND
PRF	–	PULSE REPITITION FREQUENCY
DRI	–	DELTA RI
UT	–	URINARY TRACT
PCS	–	PELVICALYCEAL SYTEM
ESWL	–	EXTRACORPOREAL SHOCKWAVE LITHOTRIPSY
AUA	–	AMERICAN UROLOGICAL ASSOCIATION
EAU	–	EUROPEAN ASSOCIATION OF UROLOGY

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INTRODUCTION

Ultrasonography remains a commonly used modality in the initial evaluation and diagnosis of renal obstruction. It can dependably detect dilatation of the urinary system proximal to the level of obstruction, which is an indirect evidence for the diagnosis. Minor dilatation of the pelvicalyceal system is a well-recognized finding in some patient with severe obstruction. Ultrasound may miss renal obstruction in a small proportion of patients in whom an obstructed pelvicalyceal system is not dilated. The obstructed pelvicalyceal system fails to dilate presumably because of low diuresis resulting from dehydration, underlying renal parenchymal disease and intermittent obstruction by calculus or decompression of the pelvicalyceal system through a tear of a calyceal fornix.

In the diagnosis of renal obstruction,the sensitivity of ultrasound is much better than its specificity. Ultrasound is less specific than excretory urography because it shows less detail of the pelvicalyceal anatomy, visualizes the dilated ureter incompletely, makes a poorer assessment of upper tract drainage and provides none of the functional information furnished by contrast medium excretion during urography.

One problem relates to the fact that ultrasound can image a fluid filled collecting system, which may not necessarily be pathological. These situations include a baggy extra-renal type of pelvis, a compound upper pole calyx and an

over-distended urinary bladder. Fluid within the collecting system is often visualized during active diuresis after an overload of oral fluids. A similar situation arises during the osmotic diuresis induced after intravenous injection of hypertonic contrast medium for urography. In addition, ultrasound visualizes a variety of dilated but non-obstructed systems, especially in reflux, or other non-obstructive causes of calyceal dilatation e.g. papillary necrosis, mega calyces, infection and residual dilatation due to previous stone or surgery.

Acute complete ureteric obstruction is associated with changes in renal blood flow as well as with an increase in renal pelvic pressure. In the first few hours, renal blood flow increases, most likely because of afferent arteriolar dilatation. After three to five hours, renal blood flow decreases, probably because of afferent arteriolar vasoconstriction produced by prostaglandins and other vasoactive substances. Decreased renal blood flow persists after 24 hours, at a time when the pressure within the collecting system is returning towards normal. The decrease in renal blood flow during obstruction can be demonstrated with Doppler ultrasound using the resistive index (RI). The time course of the RI changes is exactly as might be predicted from knowledge of the pathophysiology - increasing approximately six hours after acute calculus obstruction and remaining at its peak from 6 to 48 hours. Subsequently, the RI remains elevated but less markedly so.

AIM AND OBJECTIVES

- Intra-renal doppler indices can help identify acute renal obstruction
- Intrarenal doppler indices can help in identifying cases of renal colic rather than other causes of abdominal pain thus limiting the number of negative CT KUB.

PRIMARY AIM OF THE STUDY

- To evaluate the diagnostic utility of intrarenal arterial Doppler study in renal obstruction.

SECONDARY AIM :

- To compare intrarenal arterial doppler in patients with obstructed and non obstructed kidneys.

STUDY DESIGN

- Prospective study

INCLUSION CRITERIA

- All patients with symptoms of acute renal obstruction presenting to the emergency department within 24 hours of onset of unilateral acute renal colic were part of this analysis.

EXCLUSION CRITERIA

- a)Patients with history of
    - renal parenchymal disease
    - chronic renal obstruction
    - renal trauma
    - bilateral renal obstruction
- b) Patients on dialysis
- c) Patients having
    - a single kidney
    - congenital anomaly of the kidneys

MATERIALS & METHODS

All patients presenting to the emergency medical division within 24 hrs of onset of symptoms of unilateral acute renal colic were part of this analysis , from 2015 to 2017 are selected.

The selected patients were subjected to gray scale ultrasonography and Doppler evaluation of intrarenal arteries. Computed tomography was done for those patients in whom US could not confirm the cause of obstruction .

Evaluation by ultrasound and Doppler study

The kidney on the side of obstruction was treated as the case kidney and the contralateral normal (unobstructed) kidney served as the control.

All patients underwent USG and Doppler in Philips iU22 using C 1-5 transducer and Siemens Acuson S3000 using 6C1 HD transducer.

Presence or absence of pelvicalyceal system dilatation was assessed in each kidney on the gray-scale images.

At least three Doppler spectra were obtained from interlobar arteries along the border of the medullary pyramids and their mean was taken.

The Doppler waveforms were made using the lowest pulse repetition frequency possible without aliasing. This maximized the size of the Doppler spectrum and decreased the percentage error in the measurements. In addition, the lowest

possible wall filter for each ultrasound scanner was used. The Doppler sample width was set at 2-5 mm.

The renal RI was calculated as follows: (peak systolic velocity- end diastolic velocity)/peak systolic velocity, with the RI difference (delta RI) determined as the difference in RI of the corresponding and contralateral kidney.

Mean RI value was calculated for each kidney.

Evaluation by Computed tomography

128 slice Siemens SOMATOM Definition Edge Computerized Tomography equipment was used in this study.

Non-enhanced CT of the abdomen is performed extending from dome of diaphragm to iliac crest with 5mm spiral selections followed by 1.25 mm reconstruction. Factors given were 120 kVp and 120 mAs. CT scan was used to detect pelvicalyceal system dilatation if any, the obstructing calculus and also its site of obstruction.

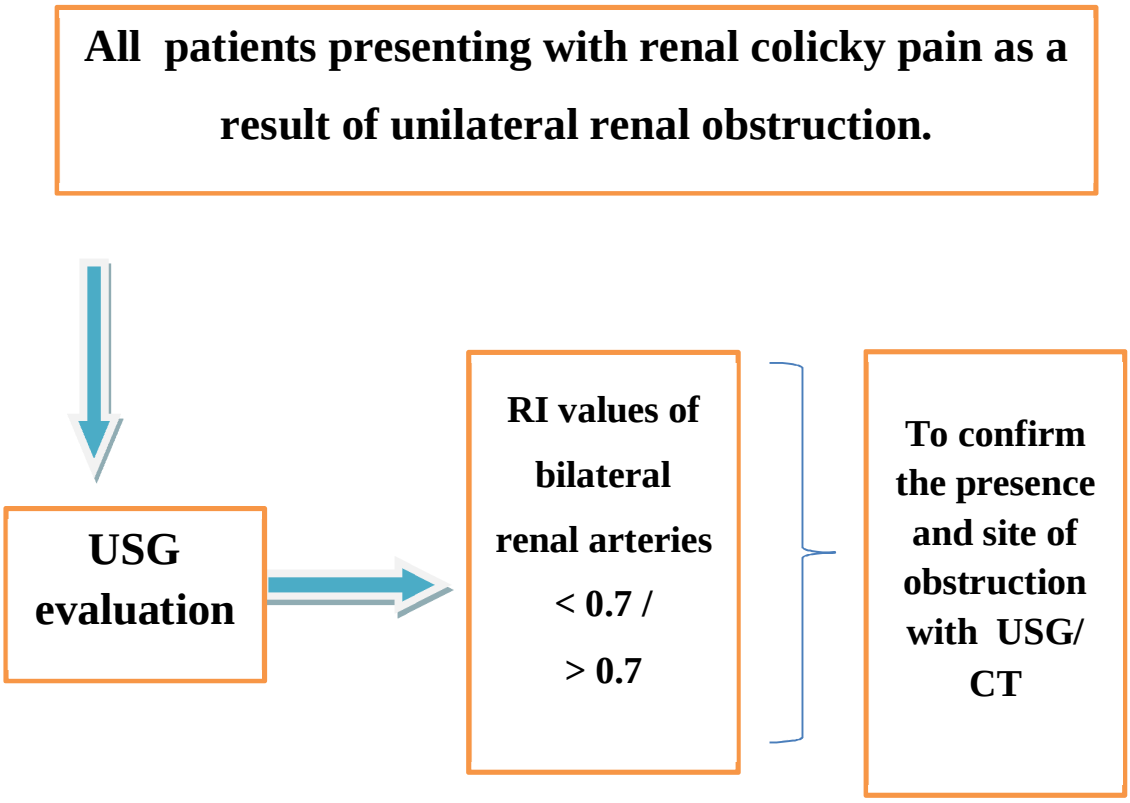
The site of obstruction was considered to be proximal, if it was up to or proximal to the L3 vertebral level and distal, if beyond.

Those patients not confirmed to have obstruction by CT were excluded from the analysis.

Statistical methods:

All data was systematically collected, tabulated and analyzed using Microsoft Excel and Strata 6 for Windows.Student t -test was used in univariate analysis for continuous variables and *Chi square* test was used for analysis of noncontiguous data. P value of less than 0.05 was considered to be statistically significant

STUDY SCHEME



RESULTS

In this study the mean Resistivity Index (RI) in segmental arteries of obstructed kidney was significantly higher than in unobstructed kidney (0.75 Vs 0.56)with a sensitivity of 85% and specificity of 93%.

The differences in RI between obstructed and unobstructed kidneys (Delta

RI) ranged from 0.09 to 0.38 with a mean Delta RI of 0.24.

CONCLUSION

Duplex Doppler ultrasound is a promising new diagnostic method in detection of acute renal obstruction especially when there is absence of collecting system dilatation.

RI measurement using Doppler US can be effectively used for the assessment of renal

colic patients by non-invasive means. It has increased sensitivity and specifivity in diagnosing acute renal obstruction when compared to conventional ultrasound and helps to avoid unnecessary CT KUB where ionizing radiation to the patient occurs.

UROLITHIASIS

Urolithiasis is a universal problem that has become increasingly prevalent in both developing and industrialized nations, with an incidence of upto 12% of men and 5% of women during their life time and also have a high rate of recurrence.

Urolithiasis have afflicted the mankind since antiquity, earliest recorded being bladder and kidney stones in Egyptian mummies dated 4800 BC.

Although the incidence of urolithiasis in developed countries has reached a plateau in recent years, it is seen to continue to increase worldwide due to variety of proposed factors, that includes obesity, dietary changes, and global warming.

Early and accurate diagnosis of obstructive uropathy allows prompt and appropriate therapy, that is essential to minimize the devastating effects of obstruction on urinary tract structure and function.

When an obstruction occurs there is an interruption to the normal urine flow at some point along the urinry tract from the renal tubule to the urethra.

Due to an obstruction there is an increased pressure within the urinary tract , causing structural and physiologic changes.

Obstructive uropathy can lead to tubular atrophy, interstitial fibrosis and inflammation, and ultimately irreversible renal injury function without appropriate timely intervention.

EMBRYOLOGY OF THE KIDNEY

The Urogenital System is developed from the intermediate mesoderm. The intermediate mesoderm forms a bulge on the posterior abdominal wall, which is called the nephrogenic cord. It extends from the cervical region to sacral region of the embryo. The pronephros is formed in the cervical region of the cord followed by the mesonephros in the thoraco-lumbar region and finally by the formation of the metanephros in the sacral region.

The human pronephros is represented by 7- 10 solid cell groups in the cervical region, which disappears at the end of 4th week of gestation. A nephric duct formed in the relation to the pronephros and ending in the cloaca, persists.

The mesonephros consists of a series of excretory tubules that develop at the thoracolumbar region. These tubules drain into the nephric duct, which is then called the mesonephric duct. Most of the mesonephric duct disappears by the end of 2^{nd} month, but some of them persists in the male, but disappear in the female.

The human kidney arises from two different sources. The excretory tubules (or nephrons) are derived from the lowest part of the nephrogenic cord, the cells of which form the metanephric blastema. The collection part of the kidney is derived from the diverticulum called ureteric bud, which arises from the lowest part of the mesonephric duct. As the ureteric bud grows cranially towards the metanephric blastema, its growing end becomes dilated to from an ampulla.

The ampulla repeatedly divides. The first three to five generations of the branches fuse to from the pelvis of the kidney. The next divisions form the minor calyces and collecting tubules. The growing tip of each of the numerous branches of the ureteric bud is dilated to form an ampulla. The cells of the metanephricblastema in contact with an ampulla undergo differentiation to form a nephron. A solid clump of cells is converted into a vesicle, which soon becomes pear shaped and open into the ampulla. The vesicle now becomes a S shaped tube, its distal end comes to be invaginated by a tuft of capillaries which form a glomerulus. The nephron is derived from this S shaped tube **(Figure 1 and 2)**

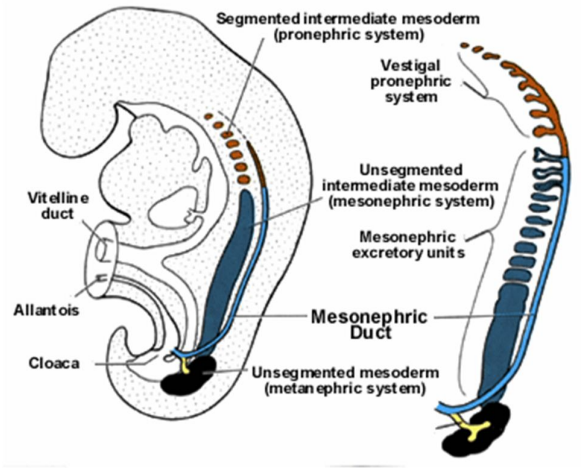


FIGURE 1: EMBRYOLOGICAL DEVELOPMENT OF KIDNEY.

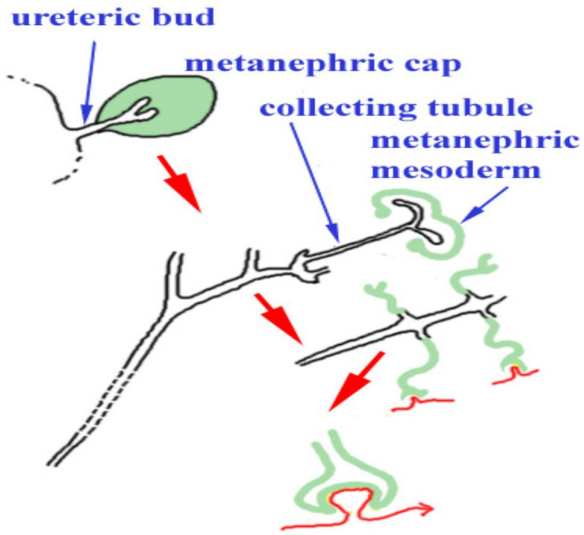


FIGURE 2: EMBRYOLOGICAL DEVELOPMENT OF NEPHRON FROM METANEPHRIC MESODERM

Ascent of the kidney:

The kidneys develop in the sacral region. In subsequent development of the embryo, differential growth of the abdominal wall causes the kidney to ascent to the lumbar region. The metanephros, at first, receives its blood supply from the lateral sacral arteries, but with its ascent, higher branch of the aorta takes over the supply. The definitive renal artery represents the lateral splanchnic branch of the aorta at the level of the 2nd lumbar segment.

Rotation of the kidney:

The hilum of the kidney, at first faces anteriorly. The organ gradually rotates, so that the hilum comes to face medially.

RENAL ANATOMY

The kidneys are also called renes from which the word ‘renal’ is derived; and nephros from which the term ‘nephron’ is derived .

Location:

The kidneys are situated in the posterior part of the abdomen, one on either side of the vertebral column, behind the peritoneum and surrounded by a mass of fat and loose areolar tissue. Their upper pole is on a level with the upper border of the twelfth thoracic vertebra, their lower pole on a level with the third lumbar. The right kidney is usually slightly lower than the left, probably on account of the vicinity of the liver. The long axis of each kidney is directed downward and lateral; the transverse axis backward and lateral.

Renal shape:

Each kidney is ovoid in outline, but it`s indented medical margins gives it a somewhat bean shaped appearance. At this concave part of each there is a vertical cleft, the renal hilum, through which the renal artery enters and renal vein and renal pelvis leave the kidney. The hilum leads into a space within the kidney called the renal sinus, which about 2.5cm in depth and is occupied by the renal pelvis, renal calyces, renal vessels and nerves and varying amount of fat.

Normal renal size:

The left kidney is slightly longer than the right one.

Normal adult renal size, plus or minus two standard deviations

Male	Right kidney	Vertical	11.3 - 14.5cm
		Width	5.3 - 7.2cm
	Left kidney	Vertical	11.6 - 14.8cm
		Width	5.3 - 7.1cm
Female	Right kidney	Vertical	10.7 - 13.7cm
		Width	4.8 - 6.6cm
	Left kidney	Vertical	11.1 - 14.3cm
		Width	5 - 6.9cm

Kidney length increases with age until approximately 20 years in both men and women and begins to diminish somewhat in the cephalocaudal dimension from about age of 20 years. In children, kidney length increases progressively with body height, and in adults similarly, kidney length is related to body height.

Covering of the kidney:

The kidneys have the following coverings:

1. Fibrous capsule: This surrounds the kidney and is closely applied to its outer surface.

2. Perirenal fat: This covers the fibrous capsule.

3. Renal fascia: This is a condensation of connective tissue that lies outside the perirenal fat and encloses the kidney and suprarenal glands; it is continuous laterally with the fascia transversalis.

4. Pararenal fat: This lies external to renal fascia. It forms a part of the retroperitoneal fat.

Relation of the kidney:

Each kidney is retroperitoneal alongside the last thoracic and upper three lumbar vertebrae, the left usually being higher than the right.

Anteriorly, the relations of the kidneys differ on two sides, expect that the anterior and medial aspects of the superior pole each kidneys are covered by the corresponding suprarenal gland.

Anterior relations of right kidney:

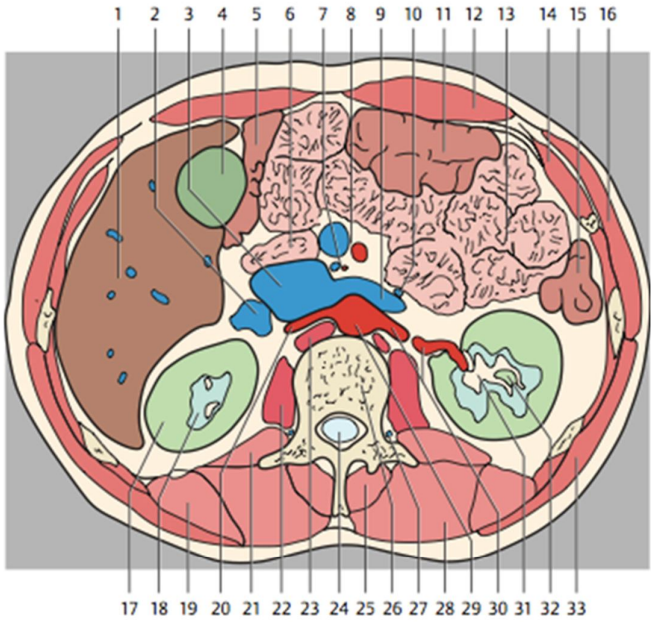
Its superior pole is related to the inferior surface of the liver. More inferiorly, the descending part of the duodenum passes across the hilum of this kidney. The right colic flexure lies anterior to its lateral border and inferior pole. Part of the small intestine lies across the inferior pole of the right kidney, it is separated from it by a film of peritoneal fluid and peritoneum.

Anterior relations of the left kidney:

The suprarenal gland caps its upper and medial portion, and the spleen borders upon its lateral aspects. The body of the pancreas with the splenic vessels lies across the kidney at or near its midsection. The left half of the transverse colon crosses the kidney below the pancreas and the descending colon overlaps its lower part laterally. A small portion of the lower pole of the left kidney is in contact with the colon, but the rest of its anterior surface is covered by the peritoneum.

Posterior relations of both the kidneys:

Each kidney lies on a muscle bed comprised of the diaphragm, the psoas major, quadratus lumborum and the transverse abdominis muscle. The diaphragm separates the upper part of the kidney from the pleura and the twelfth rib.



1. Liver
2. Right renal vein
3. Inferior vena cava
4. Gallbladder
5. Right colic flexure
6. Duodenum
7. Inferior pancreaticoduodenal artery and vein
8. Superior mesenteric artery and vein
9. Left renal vein
10. Inferior mesenteric vein
11. Transverse colon
12. Rectus abdominis muscle
13. Jejunum
14. Internal oblique muscle
15. Descending colon
16. External oblique muscle
17. Right kidney
18. Renal pyramids
19. Iliocostalis muscle, thoracic part
20. Right renal artery
21. Quadratus lumborum muscle
22. Psoas major muscle
23. Diaphragm
24. Vertebral canal
25. Spinalis muscle
26. Thoracic vertebra
27. Ascending lumbar vein
28. Longissimus thoracis muscle
29. Abdominal aorta
30. Left renal artery
31. Renal sinus
32. Renal calices
33. Latissimus dorsi muscle
34. Hepatorenal recess
35. Intercostal lymph nodes
36. Inferior duodenal recess
37. Lumbar lymph nodes
38. Greater omentum
39. Renal fascia
40. Left mesentericocolic gutter
41. Left paracolic gutter

FIGURE 3: ANATOMICAL RELATIONS OF NORMAL KIDNEY.

General Structure of Kidney

The kidney is invested by a fibrous tunica, which forms a firm, smooth covering to the organ. If a vertical section of the kidney be made from its convex to its concave border, it will be seen that the hilum expands into a central cavity, the renal sinus, which contains the upper part of the renal pelvis

and the calyces, surrounded by some fat in which are imbedded the branches of the renal vessels and nerves. The renal calyces, seven to thirteen in number, are cup-shaped tubes, each of which embraces one or more of the renal papillae. They unite to form two or three short tubes, the infundibulum and these in turn join to form a funnel-shaped sac, the renal pelvis. The renal pelvis, wide above and narrow below where it joins the ureter, is partly outside the renal sinus. The renal calyces and pelvis form the upper expanded end of the excretory duct of the kidney **(Figure 4).**

The kidney is composed of an internal medullary and an external cortical substance. The medullary substance (substantia medullaris) consists of a series of red-coloured striated conical masses, termed the renal pyramids the bases of which are directed toward the circumference of the kidney, while their apices converge toward the renal sinus, where they form prominent papillae projecting into the interior of the calyces. The cortical substance (substantia corticalis) is reddish brown in colour and soft and granular in consistence. It lies immediately beneath the fibrous tunic, arches over the bases of the pyramids, and dips in between adjacent pyramids toward the renal sinus. The parts dipping in between the pyramids are named the renal columns of Bertini, while the portions which connect the renal columns to each other and intervene between the bases of the pyramids and the fibrous tunic are called the cortical arches **(Figure4).**

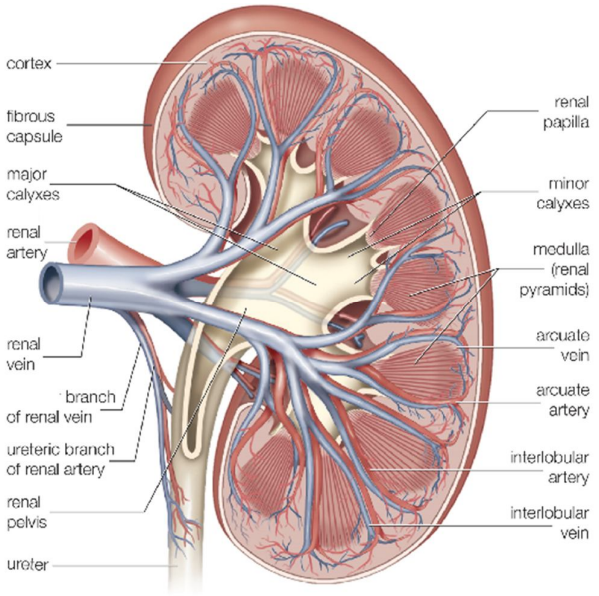


FIGURE 4: ANATOMICAL STRUCTURE OF KIDNEY

Structure of nephron

The glomerulus is a lobulated network of convoluted capillary blood vessels, held together by scanty connective tissue. This capillary network is derived from a small arterial twig, the afferent vessel, which enters the capsule and the resulting vein, the efferent vessel, emerges from the capsule at the same point. The glomerular or Bowman's capsule consists of a basement membrane, lined on its inner surface by a layer of flattened epithelial cells, which are reflected from the lining membrane on to the glomerulus. Thus between the glomerulus and the capsule a space is left, forming a cavity lined by a continuous layer of squamous cells. The renal tubules commence in the cortical substance and after pursuing a very circuitous course through the cortical and medullary substances, finally end at the apices of the renal pyramids. The tubule becomes

convoluted and pursues a considerable course in the cortical substance constituting the proximal convoluted tube. In the medullary substance, it becomes much smaller, quite straight in direction, and dips down for a variable depth into the pyramids, constituting the descending limb of Henle's loop. Bending on itself, it forms the loop of Henle and reascending, becomes suddenly enlarged, forming the ascending limb of Henle's loop and reenters the cortical substance. It ends in a convoluted tube, which resembles the proximal convoluted tubule and is called the distal convoluted tubule. This again terminates in a narrow junctional tube, which enters the straight or collecting tube. The straight or collecting tubes commence in the radiate part of the cortex. They unite at short intervals with one another so that a series of comparatively large tubes passes from the bases into the renal pyramids. In the medulla the tubes of each pyramid converge to join a central tube (duct of Bellini) which finally opens on the summit of one of the papillae; the contents of the tube are therefore discharged into one of the calyces **(Figure 5 and 6).**

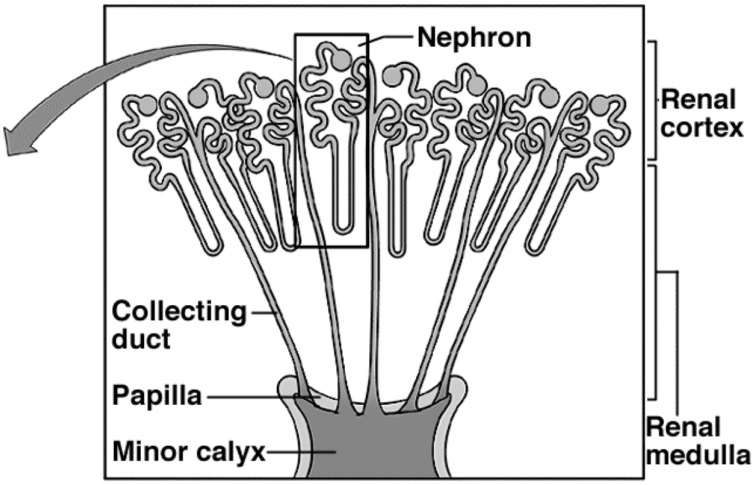


FIGURE 5: STRUCTURE OF RENAL PYRAMID

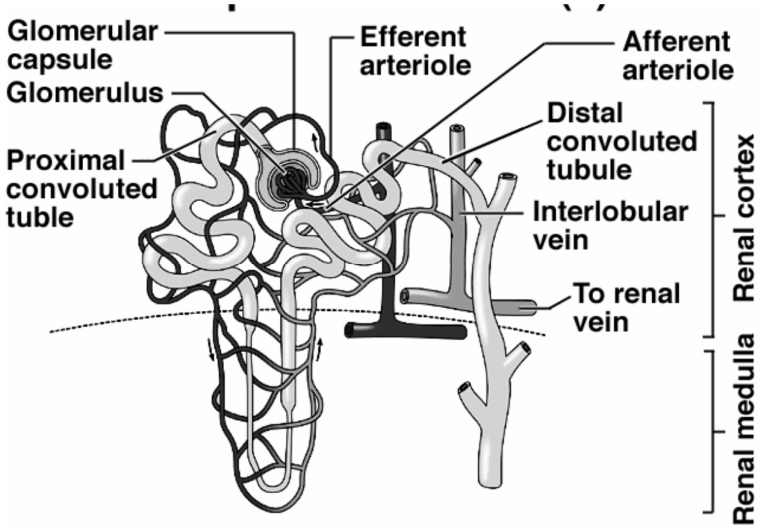


FIGURE 6: STRUCTURE OF NEPHRON

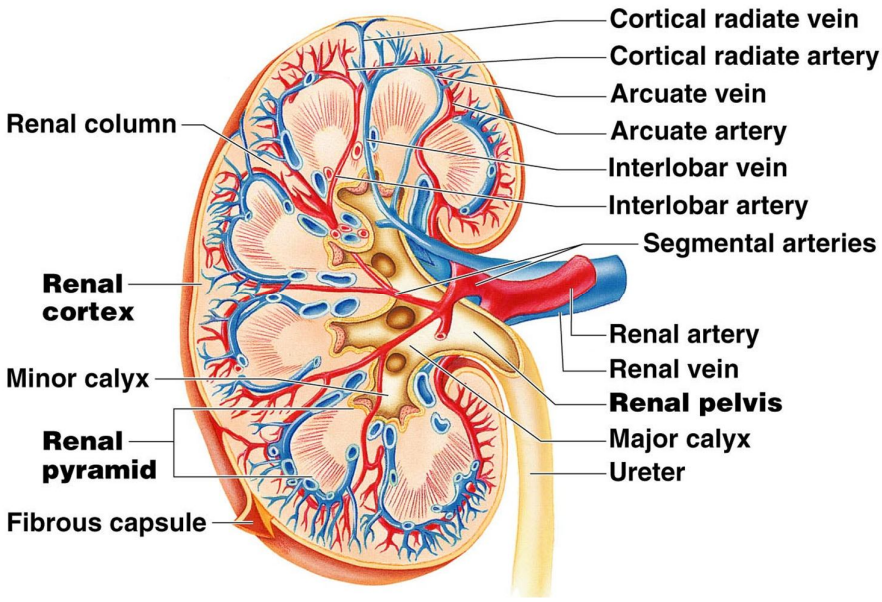


FIGURE 7: VASCULAR ANATOMY OF KIDNEY

Vascular supply of the Kidney:

Blood flow to the two kidneys is normally about 22 per cent of the cardiac output, or 1100 ml/min. The renal artery arises at right angles from the aorta, at the level of intervertebral disc between L1 and L2 vertebra immediately distal to the origin of the superior mesenteric artery. Due to the anatomical position of the abdominal aorta (slightly to the left of the midline), the right renal artery is longer, and crosses the vena cava posteriorly. Each renal artery enters the kidney via the renal hilum, dividing into segmental branches. These branches undergo further divisions to supply the renal parenchyma:

- Each segmental artery divides to form **interlobar arteries**. They are situated either side every renal pyramid.
- These interlobar arteries undergo further division to form the **arcuate arteries**.
- At 90 degrees to the arcuate arteries, the **interlobular arteries** arise.
- The interlobular arteries pass through the cortex, dividing one last time to form **afferent arterioles**.
- The afferent arterioles form a capillary network, the glomerulus, where filtration takes place. The capillaries come together to form the efferent arterioles.

In the outer two-thirds of the cortex, the efferent arterioles form what is a known as a **peritubular network**, supplying the nephron tubules with oxygen and nutrients. The inner third of the cortex and the medulla are supplied by long, straight arteries called **vasa recta**.

Venous Drainage:

The kidneys are drained of venous blood by the left and right **renal veins**. They leave the renal hilum anteriorly to the renal arteries, and empty directly into the inferior vena cava. As the vena cava lies slightly to the right, the left renal vein is longer, and travels anteriorly to the abdominal aorta.

Lymphatic drainage

Lymph from the kidney drains into the **lateral aortic nodes**, which are located at the origin of the renal arteries.

Innervation

Pacemaker cells, which are innervated by the autonomic nervous system, are located in the renal pelvis and initiate muscle contractions of the ureter with peristalsis from a cranial to a caudal direction

ANATOMY OF THE URETER

Abdominal ureter

The ureter is roughly 25-30 cm long in adults and courses down the retroperitoneum in an S curve. At the proximal end of the ureter is the renal pelvis; at the distal end is the bladder. The ureter begins at the level of the renal artery and vein posterior to these structures. This ureteropelvic junction usually coincides with the second lumbar vertebra on the left, with the right being marginally lower.

The ureter then continues anteriorly on the psoas major muscle, crossing under the gonadal vein at the level of the inferior pole of the kidney. The ureters course medial to the sacroiliac joint and then curve laterally in the pelvis. The colon and its mesentery are associated anterior to the ureters. Specifically, the

cecum, appendix, and ascending colon lie over the right ureter, and the descending and sigmoid colon lie over the left ureter.

Pelvic ureter

The ureter enters the pelvis, where it crosses anteriorly to the iliac vessels, which usually occurs at the bifurcation of the common iliac artery into the internal and external iliac arteries. Here, the ureters are within 5 cm of one another before they diverge laterally.

Before reaching the iliac vessels, the ureter passes under the gonadal vessels. The ureter crosses the iliac vessels ventrally causing middle physiologic narrowing and curves laterally in the pelvis.

In men, the lower third lies dorsally to the vas deferens, the medial umbilical ligament and superior vesical artery with the anteromedial surface of the ureter covered by peritoneum.

In women, the ureter runs posterior to the ovary and then deep to the broad ligament. The uterine artery crosses anteriorly in the rectouterine fold of the peritoneum. The transition of the ureters into the bladder causes the lower physiologic narrowing. Each ureter enters the bladder base in a tunnel-fashion diagonally through the thick muscular bladder wall, which is also angled, and therefore prevents the urine from refluxing back into the ureter when the bladder contracts.

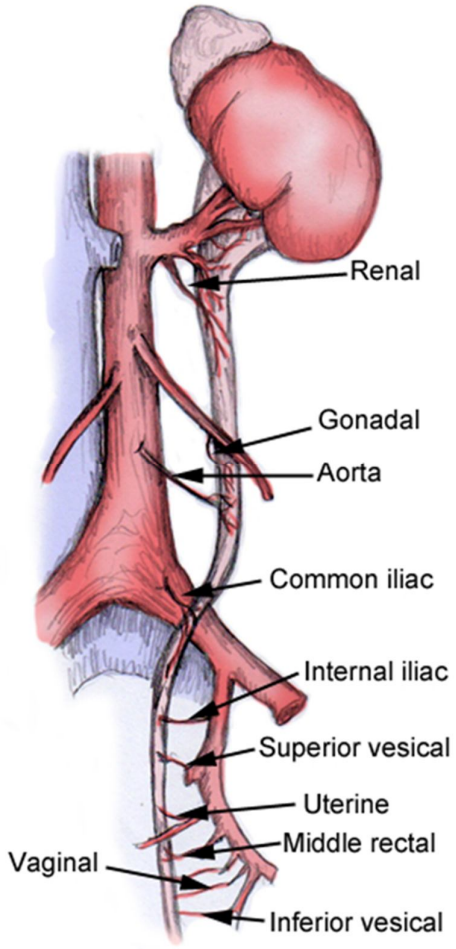


FIGURE 8: ARTERIAL SUPPLY TO THE URETER

Blood supply and lymphatic drainage

In the abdominal ureter, the arterial supply is located on the medial aspect of the ureter, whereas in the pelvis, the lateral aspect harbors the blood supply .The upper ureter is supplied by the renal artery and by branches from the gonadal artery and aorta. The arterial supply of the middle ureter is derived from the common iliac and gonadal arteries. The distal ureter is supplied by

branches of the common iliac and internal iliac branches, particularly uterine and superior vesical arteries. (**Figure 8)**

The venous drainage is paired with the arteries. Lymphatic drainage of the upper ureter joins the renal lymphatics to the lumbar nodes. The middle ureter drains to the common and internal iliac nodes. The lymphatic vessels of the pelvic ureter drain to the internal iliac and vesical nodes.

Lymphatic drainage of the upper ureter joins the renal lymphatics to the lumbar nodes. The middle ureter drains to the common and internal iliac nodes. The lymphatic vessels of the pelvic ureter drain to the internal iliac and vesical nodes

Formation of calculus

Urinary tract stone disease is likely caused by two basic phenomena.

The first phenomenon is supersaturation of the urine by stone-forming constituents, including calcium, oxalate, and uric acid. Crystals or foreign bodies can act as nidus, upon which ions from the supersaturated urine form microscopic crystalline structures. The resulting calculi give rise to symptoms when they become impacted within the ureter as they pass toward the urinary bladder.

The overwhelming majority of renal calculi contain calcium. Uric acid calculi and crystals of uric acid, with or without other contaminating ions, comprise

the bulk of the remaining minority. Other, less frequent stone types include cysteine, ammonium acid urate, xanthine, dihydroxyadenine, and various rare stones related to precipitation of medications in the urinary tract. Supersaturation of the urine is likely the underlying cause of uric and cysteine stones, but calcium-based stones (especially calcium oxalate stones) may have a more complex etiology.

The second phenomenon, which is most likely responsible for calcium oxalate stones, is deposition of stone material on a renal papillary calcium phosphate nidus, typically a Randall plaque (which are always composed of calcium phosphate) .

Calcium phosphate precipitates in the basement membrane of the thin loops of Henle, erodes into the interstitium, and then accumulates in the subepithelial space of the renal papilla. The subepithelial deposits, which have long been known as Randall plaques, eventually erode through the papillary urothelium. Stone matrix, calcium phosphate, and calcium oxalate gradually deposit on the substrate to create a urinary calculus.

Stone Classification Based on Composition

Wide range of familial, environmental, dietary, and systemic factors has seen to be contributed to the pathogenesis of renal stones . Stones are composed of a combination of crystals (both inorganic and organic) and proteins .

Calcium-based stones, which include calcium oxalate monohydrate, calcium oxalate dihydrate, and calcium phosphate stones, comprises for 70%–80% of upper urinary tract stones. Struvite stones account for 5%–15% of stones and are composed of magnesium ammonium phosphate.

Uric acid stones are unique and they can often be dissolved with urinary alkalinization accounting for 5%–10% of stones and occurs in acidic urine (pH <5.8).

	Composition	Frequency of Occurrence	KUB Radiographic Appearance	CT Appearance/Attenuation (HU)	Associated Etiologic Factors
	Calcium oxalate monohydrate and dihydrate (calcium oxalate dihydrate)	40%–60%	Radiopaque	Opacified/ 1700–2800	Underlying metabolic disorder (eg, idiopathic hypercalcuria or hyperoxaluria)
	Hydroxyapatite (calcium phosphate)	20%–60%	Radiopaque	Opacified/ 1200–1600	Usually no metabolic abnormality
	Brushite	2%–4%	Radiopaque	Opacified/ 1700–2800	. . .
	Uric acid	5%–10%	Radiolucent	Opacified/ 200–450	Idiopathic hyperuricemia or hyperuricosuria
	Struvite	5%–15%	Radiopaque	Opacified/ 600–900	Renal infection
	Cystine	1%–2.5%	Mildly opaque	Opacified/ 600–1100	Renal tubular defect

FIGURE 9: CHART ILLUSTRATING COMMONLY OCCURRING URINARY TRACT STONES AND THEIR SALIENT FEATURES.

ACUTE RENAL COLIC DUE TO CALCULUS

The colicky-type pain known as renal colic usually begins in the upper lateral midback over the costovertebral angle and occasionally subcostally. It radiates inferiorly and anteriorly toward the groin (**Figure 10).** The pain generated by renal colic is primarily caused by the dilation, stretching, and spasm caused by the acute ureteral obstruction.

In the ureter, an increase in proximal peristalsis through activation of intrinsic ureteral pacemakers may contribute to the perception of pain. Muscle spasm, increased proximal peristalsis, local inflammation, irritation, and edema at the site of obstruction may contribute to the development of pain through chemoreceptor activation and stretching of submucosal free nerve endings.

The term "renal colic" is actually a misnomer, because this pain tends to remain constant. The pattern of the pain depends on the individual’s pain threshold and perception and on the speed and degree of the changes in hydrostatic pressure within the proximal ureter and renal pelvis. Ureteral peristalsis, stone migration, and tilting or twisting of the stone with subsequent intermittent obstructions may cause exacerbation or renewal of the renal colic pain. The severity of the pain depends on the degree and site of the obstruction, not on the size of the stone.

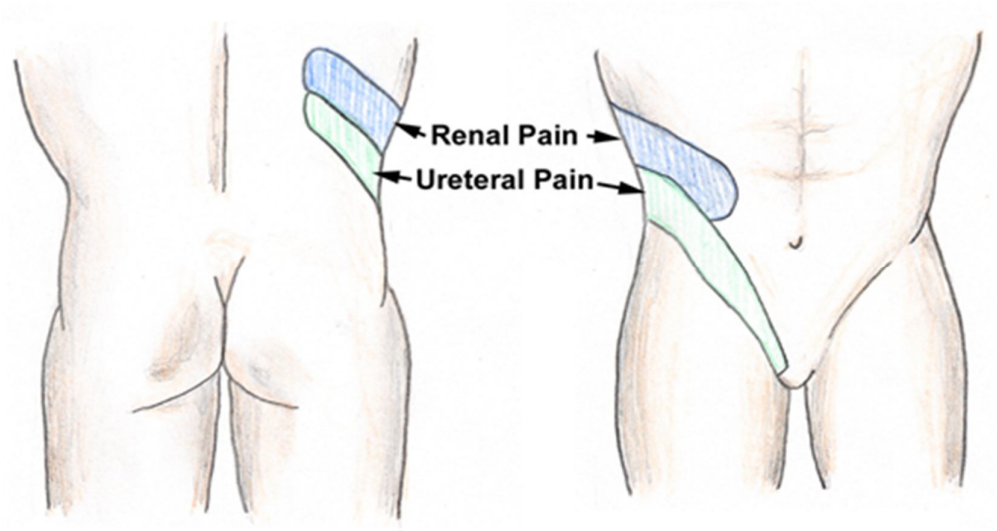


FIGURE 10: RENAL COLIC.

Most of the pain receptors of the upper urinary tract responsible for the perception of renal colic are located submucosally in the renal pelvis, calices, renal capsule, and upper ureter. Acute distention seems to be more important in the development of the pain of acute renal colic than spasm, local irritation, or ureteral hyperperistalsis.

Stimulation of the peripelvic renal capsule causes flank pain, while stimulation of the renal pelvis and calices causes typical renal colic. Mucosal irritation can be sensed in the renal pelvis to some degree by chemoreceptors, but this irritation is thought to play only a minor role in the perception of renal or ureteral colic.

Renal pain fibers are primarily preganglionic sympathetic nerves that reach spinal cord levels T-11 to L-2 through the dorsal nerve roots (**FIGURE 11 AND 12).** Aortorenal, celiac, and inferior mesenteric ganglia are also involved. Spinal transmission of renal pain signals occurs primarily through the ascending spinothalamic tracts.

In the lower ureter, pain signals are also distributed through the genitofemoral and ilioinguinal nerves. The nervi erigentes, which innervate the intramural ureter and bladder, are responsible for some of the bladder symptoms that often accompany an intramural ureteral calculus.

FIGURE 11: NERVE SUPPLY TO KIDNEY

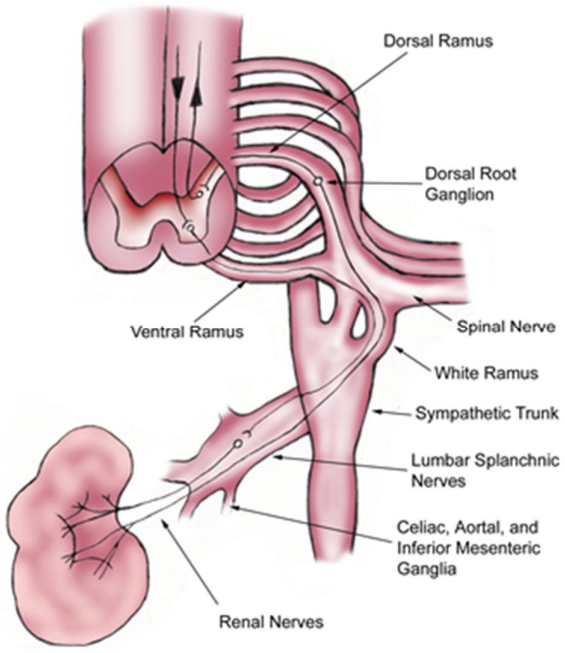


FIGURE 12: PAIN RECEPTORS

Pathophysiology of urinary obstruction

A constant obstruction, even if high grade, allows for various autoregulatory mechanisms and reflexes, interstitial renal edema, and pyelolymphatic and pyelovenous backflow to help diminish the renal pelvic hydrostatic pressure, which gradually helps reduce the pain.

The interstitial renal edema produced stretches the renal capsule, enlarges the kidney and increases renal lymphatic drainage. (Increased capillary permeability facilitates this edema.)

Distention of the renal pelvis initially stimulates ureteral hyperperistalsis, but this diminishes after 24 hours, as does renal blood flow. Peak hydrostatic renal

pelvis pressure is attained within 2-5 hours after a complete obstruction. A unique biphasic hemodynamic response is noted in complete ureteral obstruction. A short period (<2 hour) of likely prostaglandin-mediated vasodilatation occurs immediately after obstruction. After this period, renal blood flow decreases, and renal vascular resistance increases**.**

Initial studies suggested that this vasoconstriction response was primarily mechanical, due to increases in collecting system pressures. Recent research, however, suggests that complex interactions between several regulatory pathways (renin–angiotensin, kallikrein–kinin and prostaglandin–thromboxane) are responsible for intense, postobstructive renal vasoconstriction

Renal blood flow decreases to approximately 50% of normal baseline levels after 72 hours, to 30% after 1 week, to 20% after 2 weeks, and to 12% after 8 weeks.

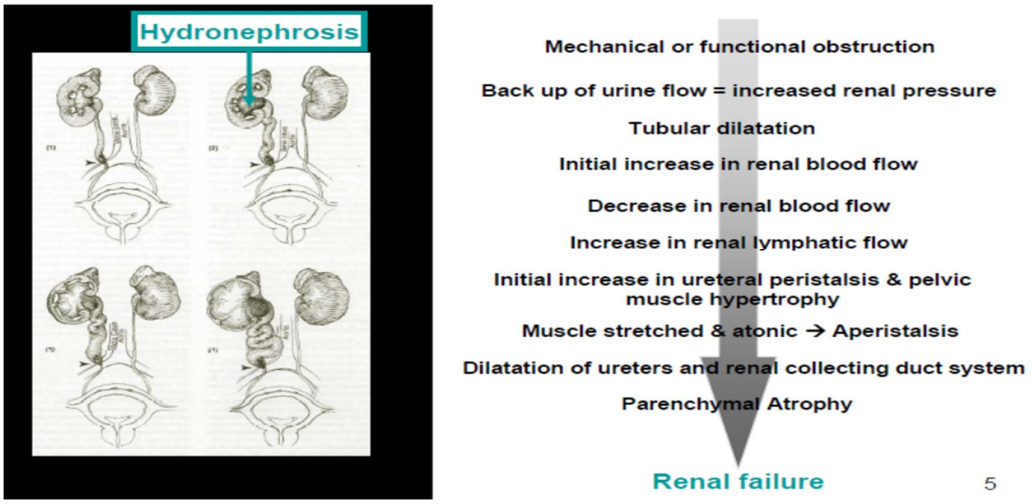


FIGURE 13: PATHOPHYSIOLOGY OF OBSTRUCTIVE UROPATHY

There are three physiologic narrowing of the ureter where stones may obstruct. The first narrowing of the ureter is the location where the renal pelvis of the kidney meets the ureter or the uretero-pelvic junction (UPJ). The next level of narrowing is where the ureter crosses the iliac vessels. At this point, the diameter of the ureter is narrowed to about 4 mm and urinary obstruction by a calculus can commonly occur. The third ureteral narrowing exist where the intramural portion of the ureter meets the bladder known as the ureteral vesico-junction, or VUJ, which measures approximately 1 - 5 mm. in size. The majority of stones become stuck at this level. Once a calculus reaches the distal ureter and approaches the bladder, symptoms of vesicle irritation are frequently noted.

Presentation and differential diagnosis :

Urolithiasis is always considered as a differential diagnosis in a patient presenting with abdominal pain. The classic presentation of renal colic is excruciating unilateral flank or lower abdominal pain of sudden onset that is not related to any precipitating event and is not relieved by postural changes or nonnarcotic medications.

With the exception of nausea and vomiting secondary to stimulation of the celiac plexus, gastrointestinal symptoms are usually absent.

Location of stone	Common symptoms
Kidney	Vague flank pain, hematuria
Proximal ureter	Renal colic, flank pain, upper abdominal pain.
Middle section of ureter	Renal colic, anterior abdominal pain, flank pain
Distal ureter	Renal colic, dysuria, urinary frequency, anterior abdominal pain, flank pain

TABLE 1: COMMON SYMPTOMS BASED ON THE STONE LOCATION

Calculous disease have also been encountered in asymptomatic patients and found incidentally on imaging studies or during the evaluation of microhematuria. Symptoms similar to those of renal colic can be caused by noncalculus conditions.

In women, gynecologic manifestations that have to be considered include ovarian torsion, ovarian cyst and ectopic pregnancy.

In men, symptoms of testicular processes, such as a tumor, epididymitis or prostatitis, may mimic the symptoms of distal ureteral stones. Other general causes of abdominal pain, such as appendicitis, cholecystitis, diverticulitis, colitis, constipation, hernias or even arterial aneurysms, should be ruled out.

Symptoms mimicking those of urolithiasis occur with urologic lesions like congenital ureteropelvic junction obstruction, renal or ureteral tumors, and other causes of ureteral obstruction.

Confirmation of the diagnosis:

The diagnosis of urinary tract calculi begins with a focused history. Key elements include past or family history of calculi, duration and evolution of symptoms, and signs or symptoms of sepsis. The physical examination has been more valuable for ruling out nonurologic disease. Urinalysis is to be performed in all patients with suspected calculi.

Apart from the typical microhematuria, important findings to consider are the urine pH and the presence of crystals, which can help to identify the stone composition.

Patients with uric acid stones usually present with an acidic urine, and those with stone formation resulting from infection have an alkaline urine. Identification of bacteria is important in planning therapy, and a urine culture is to be routinely performed. Limited pyuria is a fairly common response to irritation caused by a stone and, in absence of bacteriuria, is not generally indicative of co- existent urinary tract infection.

Symptomatic stones present as abdominal pain. Renal colic can be suspected based on the history and physical examination, but diagnostic imaging is essential to confirm or exclude the presence of urolithiasis.

Diagnostic imaging is essential to confirm the size and location of urinary tract calculi. A diagnosis of renal colic cannot be based on the clinical findings alone.

Below is a brief discussion regarding the various imaging modalities available for diagnosing urolithiasis causing acute unilateral renal obstruction, by using the ALARA principle.

Imaging of urolithiasis has evolved over the years due to technologic advances and a better understanding of the disease process.

Plain abdominal radiograph has very low sensitivity for detection of urolithiasis.

Gray scale ultrasound may reveal a stone in the collecting system or it can suggest an obstructive uropathy by demonstrating hydronephrosis., dilatation of pelvicalyceal system and ureter.

In early obstruction study can be equivocal without any significant dilatation of the collecting system. And it does not provide functional data regarding obstruction.

Intrarenal duplex Doppler ultrasonography is been proposed as a non-invasive, test to distinguish between an obstructed and non-obstructed kidney, with resistive index (RI) and delta resistive index (delta RI), variable most widely measured.

This study is carried out to evaluate the efficacy of Doppler ultrasound in the diagnosis of acute unilateral renal obstruction in patients presenting to the Emergency department with acute flank pain.

IMAGING OF THE URINARY TRACT

Intravenous urography carried the risk of exposure of the subjects to ionizing radiation, as well as to complications related to contrast media injection.

Non enhanced helical CT is considered the prime radiological study for evaluating urolithiasis and is the gold standard, having a sensitivity of 95% and specificity of 98%, the draw backs like high cost, ionising radiation to the patient , Ultrasound is modified to enhance its diagnostic performance for detection of obstructive uropathy and Doppler US is used to overcome this problem.

Conventional imaging techniques:

Plain abdominal radiography (KUB), intravenous urography (IVU), standard renal sonography and CT KUB are the usual imaging modalities for evaluating acute renal colic.

Conventional abdominal radiography (KUB)

Plain abdominal radiography (KUB radiography) is useful for assessing total stone burden, as well as the size, shape, composition, and location of urinary calculi in some patients. Calcium-containing stones (approximately 85% of all

upper urinary tract calculi) are radiopaque, but pure uric acid, indinavir-induced, and cystine calculi are relatively radiolucent on plain radiography.

When used with other imaging studies, like renal ultrasonography or CT scanning, the plain film helps provide a better understanding of the characteristics of urinary stones revealed with these other imaging studies. They also help in planning surgical therapy.

Not all urinary calculi are visible on the KUB radiograph, because of their small size, stone radiolucency, or overlying gas, stool, or bone. The stones that are observed can be correlated with opacities found on other studies for identification and tracking progress.

If a stone is not visible on a flat plate radiograph, it could be a radiolucent uric acid stone. Such a stone is more likely if the urine pH indicates very acidic urine. In practice, any patient with symptoms of acute renal colic who demonstrates a urine pH lower than 6.0 should be considered at risk for a possible uric acid stone. If a stone of adequate size is visible on a CT scan but not visible on KUB, then uric stones have to be considered.

The flat plate radiograph is inexpensive, quick, and usually helpful even if no specific stone is observed. It is extremely useful in following the progress of previously documented radiopaque calculi and checking the position of any indwelling double-J stents. The KUB radiograph can suggest the fluoroscopic

appearance of a stone, which determines whether it can be targeted with ESWL.

The KUB radiograph is also quite accurate for helping determine the exact size and shape of a visible radiopaque stone and sometimes is more accurate than CT in this regard. Note that most stones will appear larger on KUB radiograph than on CT, with CT-based measurement of maximum stone dimension approximately 12% smaller than a corresponding KUB-based measurement.

Many calcifications observed on the KUB radiograph are phleboliths, vascular calcifications, calcified lymph nodes, appendicoliths, granulomas, various calcified masses, or even bowel contents. All of these are confused with urinary tract calculi.

Plain radiography for the evaluation of nephrolithiasis has several limitations that include bowel gas, extrarenal calcification and large patient habitus, diminishing the sensitivity of x-ray KUB.

KUB plain film radiography and fluoroscopy use a single energy source to produce photons, which pass through tissues in an anterior-to-posterior orientation encountering a contralateral receiver. This technique uses the same fundamental concepts as CT but in a single plane.

Historically, KUB radiography has been used to conduct an intravenous pyelogram, which enabled evaluation of the presence of hydronephrosis and obstruction. This modality is been largely replaced with the introduction of CT

as an imaging technique. Plain radiography relatively has low sensitivity (40-50%) and specificity for renal and ureteral calculi. Many patients have numerous pelvic calcifications that make pinpointing specific stones difficult. Any calcific density observed on a KUB radiograph that happens to overlie the course of the ureter is not guaranteed to be a stone.

Advantages of KUB radiography include relatively low ionizing radiation exposure compared with CT (0.15 mSv) and low cost .This imaging modality only views stones at one angle, thereby decreasing the accuracy and reduced sensitivity and specificity , therefore, limiting its utility.

Many stone types can be visualized using KUB radiography; however cystine and struvite stones often are poorly visible on KUB radiography, and uric acid and matrix stones are not visible at all. Xray KUB has a low sensitivity of 37% for stones <5mm.

To overcome this phenomenon, ultrasonography and KUB radiography can be performed in conjunction, enabling the higher sensitivity of ultrasonography to augment the higher specificity of KUB radiography. The AUA suggests use of this combined imaging approach for the evaluation of ureteral stone disease during stone passage or after treatment.

For example, an obstructing stone can be observed to move after treatment. However, small stones over a bony structure, or those shadowed by bowel gas can be concealed.

FIGURE 14: X-RAY DEMONSTRATING LEFT URETERIC CALCULUS

FIGURE 15: INTRAVENOUS PYELOGRAM (IVP) DEMONSTRATING DILATION OF THE RIGHT RENAL COLLECTING SYSTEM AND RIGHT URETER CONSISTENT WITH RIGHT URETEROVESICAL STONE.

Intravenous pyelography :

Intravenous pyelography was been considered the standard imaging modality for uri nary tract calculi previously.

The IVP provides useful information about the stone (size, location, radiodensity) and its environment (calyceal anatomy, degree of obstruction), as well as the contralateral renal unit (function, anomalies).

The accuracy of intravenous pyelography can be maximized with proper bowel preparation, and the adverse renal effects of contrast media may be minimized by ensuring that the patient is well hydrated.

Unfortunately, these preparatory steps require time and often cannot be accomplished when a patient presents in an emergency situation. Intravenous pyelography has greater sensitivity (64 to 87 per- cent) and specificity (92 to 94 percent) for the detection of renal calculi.

The limitation of IVP is that it can be confusing in the presence of nonobstructing radiolucent stones, which may not always generate a filling defect.

Furthermore, in patients with high-grade obstruction, even prolonged reimaging at 12 to 24 hours may not demonstrate the level of obstruction because of inadequate concentration of the contrast medium.

The contrast media used in IVP carry the potential for adverse effects like nephrotoxic effect. Serum creatinine levels must be measured before contrast media are administered. Adverse effects like metabolic acidosis in diabetes patients on metformin. Other factors like presence of ionising radiation to the patients, especially in follow up cases are major draw backs of this modality.

CT KUB:

Noncontrast helical CT is fast and accurate, and it readily identifies all stone types in all locations. Its sensitivity (95 to 100 percent) and specificity (94 to 96 percent) suggest that it may definitively exclude stones in patients with abdominal pain. Associated signs, such as renal enlargement, perinephric or periureteral inflammation or “stranding,” and distension of the collecting system or ureter, are sensitive indicators of the degree of ureteral obstruction.

Hounsfield density of calculi may be used to distinguish cystine and uric acid stones from calcium-bearing stones and is capable of further subtyping the calcium stones into calcium phosphate, calcium oxalate monohydrate and calcium oxalate dihydrate stones. Noncontrast helical CT is also useful in diagnosing nonurologic causes of abdominal pain, such as abdominal aortic aneurysms and cholelithiasis. The estimated sizes of renal calculi deter- mined using this imaging technique vary slightly from those obtained with KUB radiography.

Draw backs for this modality are they are generally more expensive than other modalities like Duplex Doppler US, Xray KUB & IVP and the burden of radiation.

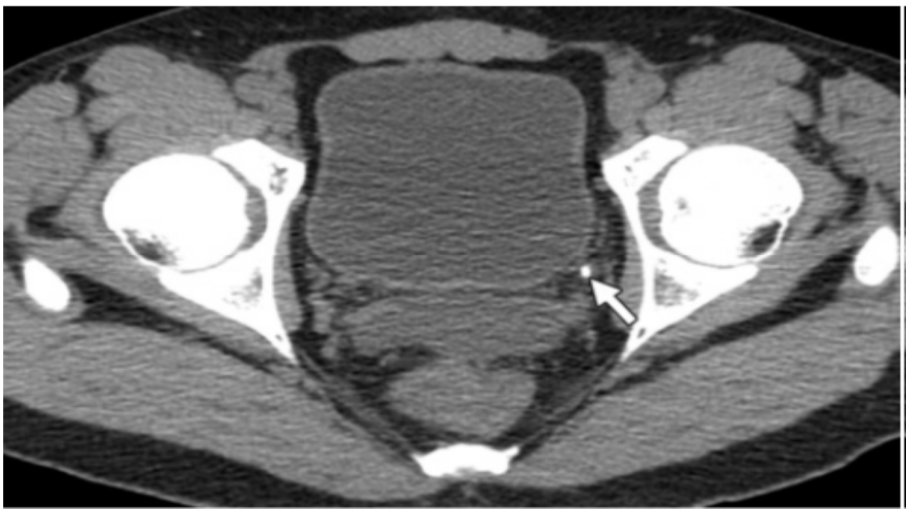


FIGURE 16: URETERAL STONE IN A 45-YEAR-OLD MAN WHO HAD PRESENTED WITH ACUTE LEFT FLANK PAIN. AXIAL UNENHANCED MULTIDETECTOR CT SCAN SHOWS A 4-MM STONE IN THE LEFT DISTAL URETER (ARROW).

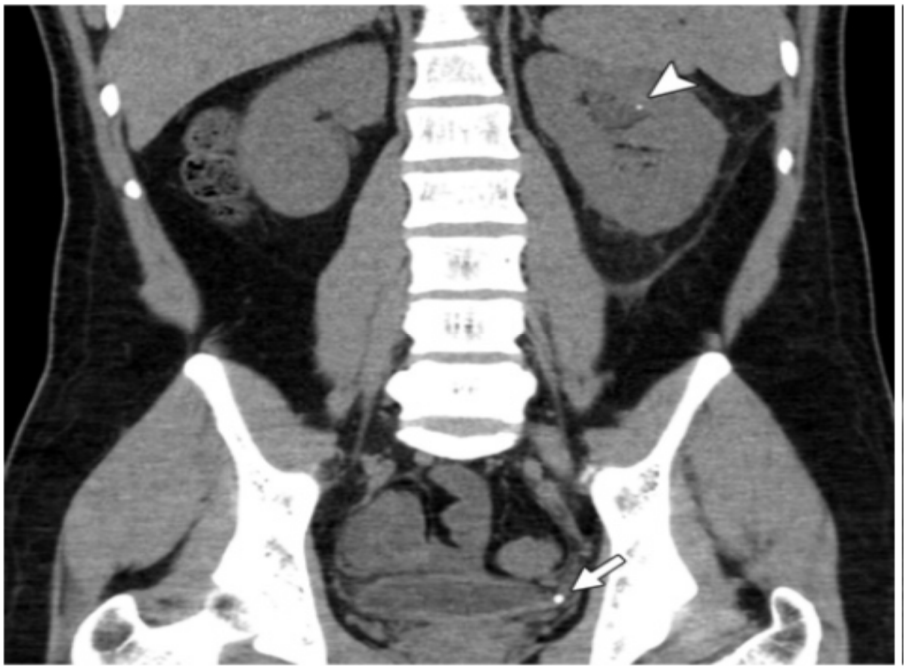


FIGURE 17 : CORONAL REFORMATTED IMAGE SHOWS THE LEFT DISTAL URETERAL STONE (ARROW), ALONG WITH A 2-MM STONE IN THE LEFT UPPER POLE (ARROWHEAD) THAT WAS NOT SEEN ON THE AXIAL IMAGES.

Radiation Dose

Despite the immense benefits of multidetector CT, a key concern regarding its use in stone disease is the risk of radiation exposure. This is particularly true in young individuals who undergo repeated CT examinations due to recurrent stone disease and are consequently likely to be at risk for greater cumulative lifetime exposure.

The effective radiation doses with unenhanced CT range from 2.8 to 13.1 mSv for men and from 4.5 to 18 mSv for women, all of which are higher than with excretory uroraphy (1.5 mSv for three-film and 2.1 mSv for six-film excretory urography) Numerous practical approaches for dose reduction in CT for urolithiasis have been advocated in the literature .

In spite of the continuing efforts to diminish radiation dose, caution is necessary to avoid “going overboard” with dose-reduction techniques. Low-dose CT can potentially degrade image quality in obese patients, and there remains a possibility of missing alternative diagnoses in selected patients with suspected urinary calculi.

Ultrasonography:

Ultrasonography is an excellent modality for evaluation of the renal collecting systems, the renal parenchyma, and the bladder but poor visualization of the ureters, especially in patients with a large amount of bowel gas or sub-cutaneous fat. Gray-scale US is less sensitive than CT for the detection of intrarenal calculi, but highly sensitive for the detection of ureteral obstruction in the setting of acute flank pain.

It is a reasonable modality used for surveillance of patients with known stone disease.

It is the test of choice in pregnant patients due to its lack of ionizing radiation.

HISTORY OF ULTRASOUND & DOPPLER

The basis of ultrasound- the Piezoelectric Effect dates back as early as in 1880

The Curie brothers, Pierre and Jacques Curie first demonstrated the Piezo-electric effect in 1880.

Golton in 1883 was the first to produce sound frequencies up to 25000 cycles per second or Hertz (Hz), much above the range of human hearing.

The technology of producing ultrasound and the characteristics of sonic waves have been known for many years. The first major attempt at a practical application was made in the unsuccessful search for the sunken Titanic in the North Atlantic in 1912.

Paul Langevin was the first to use pulse echo technique in the detection of submarines.

SONAR (Sound Navigation And Ranging) was the first important successful application.

The first medical use of ultrasound was made by Karl Kursik in 1942, who attempted to locate brain tumors using two opposing ultrasound transducers

The first step towards making an anatomical image with ultrasound was made by Karl Dussick and Fredrick Dussick in 1947.

Donald, I. first presented the demonstration of tissue interfaces within the body by ultrasonic waves in 1961.

Christian Johann Doppler (29.11.1803 – 17.03.1853), Professor of practical geometry and elementary mathematics at the State Technical Academy in Prague was the first person to propose "The theory of Doppler effect". In his work, Doppler postulated his principle (later coined the Doppler Effect) that the observed frequency of a wave depends on the relative speed of the source and the observer, and he tried to use this concept for explaining the colour of binary stars.

The Doppler effect of sound was verified by Buys Ballot in 1845.

Doppler ultrasound was developed by Satomura (Osaka University) in 1955.

Howry, D. in 1965 gave a brief atlas of ultrasonic radiologic results.

Sonographic characteristics of normal kidney:

The normal renal ultrasound size has been reported to be 10 cm in length, 5 cm wide and 2.5 cm thick. Renal size is related to sex, age and built of the patient.

Brandt T. D et al stated that the ultrasound dimensions are smaller than those noted by radiography because there is neither geometric magnification nor change in the size related to an osmotic diuresis from contrast. In prone position, the right mean length was found to be 10.74cm (±1.35 standard

deviation). The kidney measures approximately 5.0cm to 5.5cm in width and approximately 3.5cm to 4 cm in depth.

The length of the normal adult kidney is usually between 10-12 cm but there is wide range of 7 to 14 cm in patients with normal renal functions.

Ninan V.T et al conducted a comparative study of methods of estimating renal size in normal adults, compared the actually bipolar length which was measured during donor nephrectomy in one hundred live related voluntary kidney donors, with the renal bipolar length estimated from abdominal sonogram, abdominal plain x- ray, intravenous pyleogram and renal angiogram. Ultrasound was found to measure the kidney accurately (mean difference between estimated size and actual = -3.4 mm + SD 0.96), than plain X-ray (mean difference from actual 13mm + SD 5.24), IVP (mean difference from actual 16.9 mm + SD 5.74), and renal angiogram (mean difference from actual 15.2 mm + SD 5.77). The mean ultrasound measurements of the right and left kidneys found in their study were 9.52 + Standard deviation 0.62cm on the right side and 9.55 cm + Standard deviation0.65 on the left side.

The renal margin is normally smooth and sharply defined, due to renal capsule, but the inner margins of the parenchyma adjacent to the echoes from the sinus fat, is less well defined.

Sonographically, the kidney is divided into the peripheral parenchyma, which includes cortex and medulla and the central renal sinus. The renal cortex

generates low level echoes that classically are described as less intense than the echogenicity of the liver, spleen or renal sinus.

The renal medulla is visualised sonographically as triangular hypoechoic renal pyramids, containing collecting tubules, located inside the cortex. The medulla is sonolucent. The cortex and medulla can be differentiated in approximately half of the adult patients depending on the patient's body habitus and frequency of the transducers used.

Corticomedullary differentiation is best appreciated in thin patients with a high frequency transducer eg.5MHz. The detectability of the medulla also changes with patient's hydration, becoming more prominent with diuresis. The intense central echoes of the renal sinus are predominantly due to hilar adipose tissue with fibrous septae, blood vessels and lymphatics as secondary contributors.

Splitting or discontinuity of central echo complex is an early sign of hydronephrosis due to obstruction. Based on the nephrotomographic appearance suggesting renal sinus lipomatosis, fat has been implicated in creating an ultrasonic appearance similar to hydronephrosis. Hence anechoic renal fat is considered one of the many causes of false positives for hydronephrosis on renal ultrasound examinations.

Organ echogenicity from greatest to least in the normal patient is as follows: Renal sinus, pancreas, liver, spleen, renal cortex, renal medullary pyramids.

With aging, the kidney becomes relatively wider and thicker with age, as judged from the renal shape index. One possible explanation for this phenomenon could be the relaxation of the abdominal wall with age, so that the kidneys are squeezed less in older persons. This would also explain the broadening that becomes most pronounced for the right kidney, which has been squeezed more because of the liver. The parenchyma normally thins with age and part of this is due to the development of the sinus lipomatosis where the amount of fat in the renal sinus is increased.

In neonates, the renal cortical echogenicity is equal to the echogenicity of the liver or spleen and the corticomedullary junction and pyramids are more prominent than in adults.

The medulla comprises a large volume in the neonate compared with the adult, and prominent echolucent pyramids are characteristic. The neonatal kidney demonstrates a paucity of renal sinus echoes due to minimal renal sinus fat.

Within 2-6 months, the kidneys become progressivley less echogenic than the liver and assume the features of the adult kidney between 6-24 months of life.

There are few anatomical variants that are commonly encountered with renal sonography. One variant is the column of Bertin, in which part of the renal cortical tissue projects into the renal sinus. These pseudomasses of normal renal parenchyma are most commonly at the junction of upper and middle thirds of the kidney, more frequently on the left.

Lafortune M et al reported that columns are evident sonographically in 47% of the healthy subjects with bilaterality in 18% and 4% incidence of two columns in one kidney. The projections into the renal sinus are also always isoechoic with the cortex, and the renal contour remains smooth.

Scanning Technique: Proper scanning technique was adviced by many authors for better evaluation of the kidneys.

Sanders R.C, et al demonstrated that the kidneys are best examined in prone position unless the patient is pregnant or too ill to tolerate this position and full bladder is not necessary. Patient should be made as comfortable as possible. Although the kidneys lie close to the patient's back they flop forward a variable distance when patient is in prone position; so output and time gain compensation cannot be pre-set. So variability in renal axis and lateral location is bound to intervene. Hence, transverse scan is advocated first before the longitudinal scan.

The location of the upper pole of kidney within the ribcage is often difficult in prone position. Hence, right kidney is best studied with the patient supine, utilising liver as an acoustic window. The best image of the left kidney is obtained with the patient in right lateral decubitus position with the arm extended over the head, using spleen as an acoustic window.

Standard renal sonography can appear normal in 50% of patients with acute urinary obstruction. Distinguishing an obstructed from a non-obstructed dilated

renal collecting system may be difficult particularly in the absence of visualization of an obstructing agent..

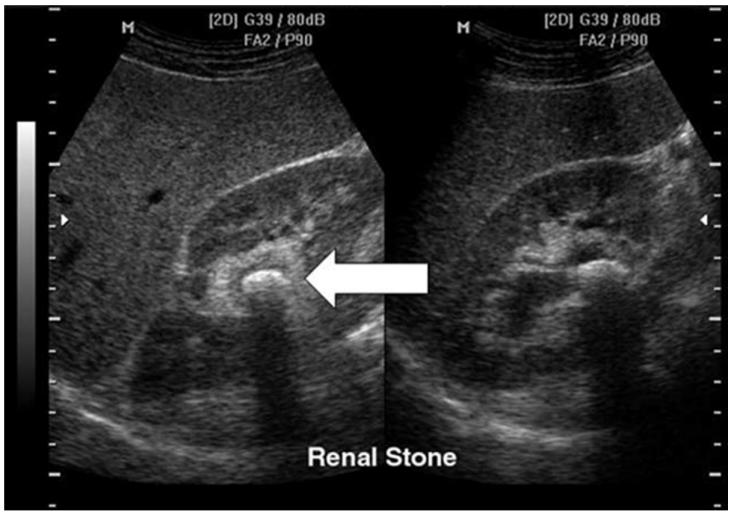


FIGURE 18 : B MODE US SHOWING A CALCULUS WITH POSTERIOR ACOUSTIC SHADOWING

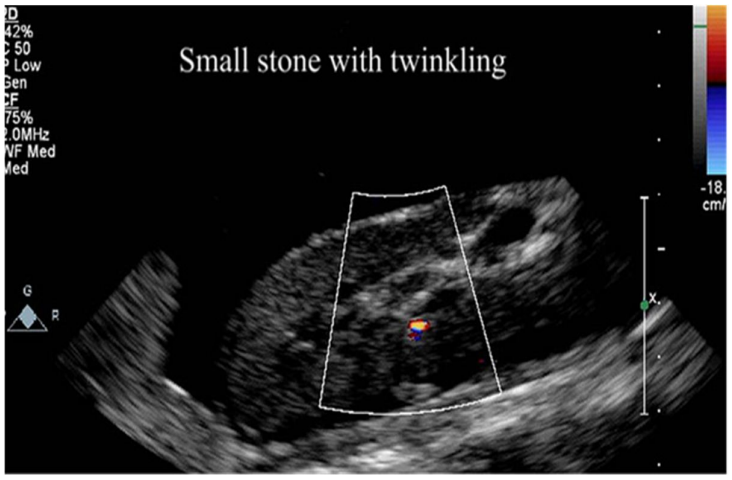


FIGURE 19 : B MODE US DEMONSTRATING A CALCULUS WITH THE TWINKLING ARTEFACT.

Poor visibility of the lumbar portion of the ureter represents has been a major limitation of renal ultrasonography.

Gray scale sonography is a commonly used modality in the initial evaluation and diagnosis of renal obstruction. Most of the time gray scale sonography detects a calculus with posterior acoustic shadowing and dilatation of the urinary system proximal to the level of obstruction, providing an indirect evidence for the diagnosis.

B-mode ultrasonography makes use of physical differences between stones and surrounding tissues to detect the stones. Relative to soft tissues, stones strongly reflect ultrasonic waves and appear as bright echogenic structures in the ultrasonographic image. Ultrasonic waves are unable to penetrate through stones, leaving a nonechogenic shadow beyond the stone in the image.

Hydronephrosis is defined as dilatation of the drainage system of the kidney, i.e., the calices, the infundibula, and the pelvis , this is demonstrated in B mode ultrasound with a sensitivity of 90% but less specific 65%- 84% in the diagnosis of renal obstruction.

While the diagnosis of hydronephrosis is usually based on conventional gray-scale US, ancillary ultrasonographic techniques may be helpful for further evaluation.

B mode USG was able to detect obstruction with a sensitivity of 82.5% and specificity of 92.5%, Hydronephrosis was detected with a sensitivity of 97.5% and a false rate of 2.5%.

GRADING OF HYDRONEPHROSIS :

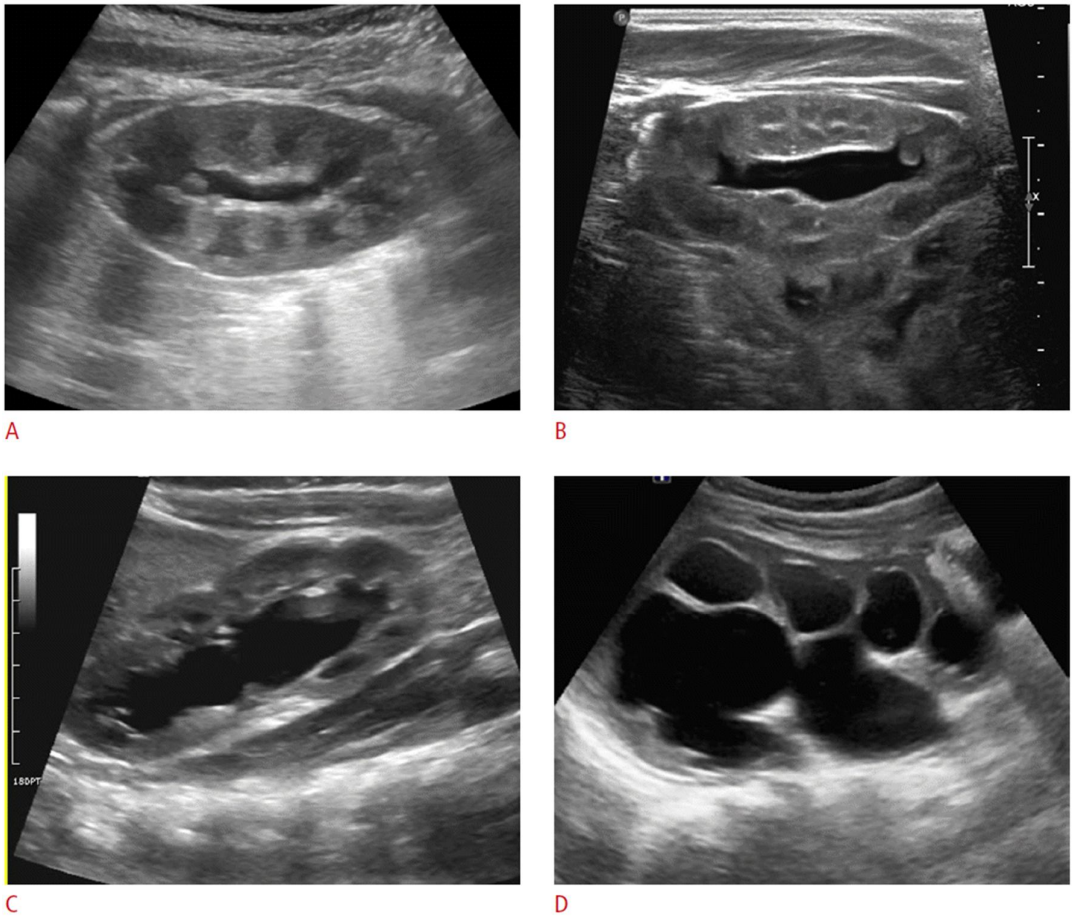


FIGURE 20 : B MODE IMAGES SHOWING VARIOUS GRADES OF HYDRONEPHROSIS

A. GRADE 1: renal pelvis is mildly dilated without calyceal dilation.

B. GRADE 2: renal pelvis is further dilated and some calyces may be visualized.

C. GRADE 3: renal pelvis and minor calyces are diffusely dilated, but renal parenchyma is of normal thickness.

D. GRADE 4: as for grade 3, but with thinning of the renal parenchyma over the dilated calyces.

CDUS adds additional value over gray scale US in evaluation of urolithiasis.

The sonographic twinkling artefact that is defined as rapidly altrnating CD signal seen deep to a strong reflector when imaging is done with high PRF, there by increasing the sensitivity of US for diagnosing urolithiaisis.

Doppler US also help to differentiate renal hilar vasculature from a dilated renal collecting system occuring in hydronephrosis.

CDUS also demonstrates the lack of a ureteral jet in case of obstruction.. Ultrasonography can be adequate for acute management in the emergency department, clear advantages of ultrasonography includes easy availability, cost and lack of exposure to ionizing radiation.

Ultrasonography is unique in that it is the only portable imaging modality for evaluation of nephrolithiasis allowing practitioners to evaluate patients at the

bedside. Ultrasonography have also traditionally been considered to cost about half as much as a standard CT.

The effects of ionizing radiation accumulate throughout a patient's life, therefore, exposure must be as low as reasonably achievable, especially in paediatric patients (<14 years old) or those who are pregnant to prevent cumulative exposure to levels of radiation that are able to induce malignancies.

In pregnant patients concern also exists regarding the potential teratogenic effects of radiation on the developing fetus.

Ultrasonography does not use ionizing radiation, for this reason the AUA, EAU and ACR recommend that ultrasonography be the first-line imaging modality in young and pregnant patients.

Ultrasonography also has increased accuracy in children owing to their small body size meaning that the distance between the ultrasonography probe and anatomy of interest is reduced.

Pulsed Doppler examination of the intrarenal renal artery can differentiate between obstructive and non-obstructive hydronephrosis. The diagnosis of obstructive uropathy can be missed by USG as pyelocaliectasis occur late in obstructive conditions and often the findings are normal despite severe renal dysfunction.

Various study show that B mode USG was able to detect obstruction with a sensitivity of 82.5% and specificity of 92.5%, Hydronephrosis was detected with a sensitivity of 97.5% and a false rate of 2.5%.

Further advancement to conventional USG is Doppler duplex USG, that allows the determination of RI. In renal obstruction there is increased renal vascular resistance leading to drop in diastolic flow, predominant change in Doppler wave form.

Duplex Doppler thus helps in differentiating obstructive and non obstructive uropathy.

Renal Doppler sonography

Animal studies have shown that there is a definite rise in vascular resistance in the renal arteries when the kidney is obstructed. Doppler ultrasonography enables detection of subtle intrarenal blood flow changes associated with various pathophysiologic conditions.

Duplex and colour Doppler imaging are able to demonstrate both normal and abnormal renal blood flow. Normal flow within the renal artery and its branches has a “low resistance” perfusion pattern, with continuous forward blood flow during diastole. The most common parameter used to describe changes in Doppler arterial spectra is the resistive index (RI) ([peak systolic velocity – end diastolic velocity] / peak systolic velocity). RI is an easily calculated, angle-independent measurement.

Technique:

Most studies describing the potential use of Doppler sonography for evaluating renal disease have stressed the need for meticulous technique**.** The highest frequency probe that gives measurable waveforms should be used, supplemented by color or power Doppler sonography as necessary for vessel localization. Arcuate arteries (at the corticomedullary junction) or interlobar arteries (adjacent to medullary pyramids) are then insonated using a 2 to 4 mm Doppler gate. Waveforms should be optimized for measurement using the lowest pulse repetition frequency without aliasing (to maximize waveform size), the highest gain without obscuring background noise, and the lowest wall filter. Three to five reproducible waveforms from each kidney are obtained, and RIs from these waveforms are averaged to arrive at mean RI values for each kidney.

Several studies have shown that a normal mean renal RI is approximately 0.60. The largest series to date (58 patients) reported a mean (±SD) RI of 0.60 ± 0.01 for subjects without preexisting renal disease.

Three prior studies also reported normal mean RI values of 0.64 ± 0.05 (21 patients)**,** 0.58 ± 0.05 (109 kidneys) and 0.62 ± 0.04 (28 patients)**.** In general, most sonographers now consider 0.70 to be the upper threshold of the normal RI in adult.

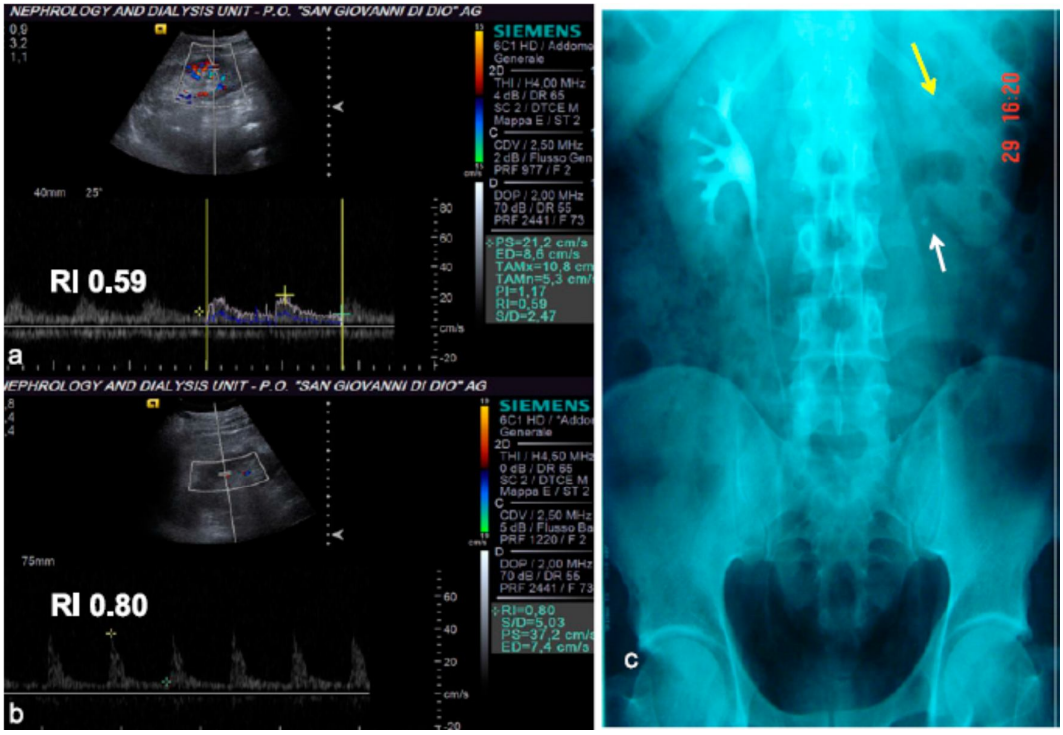


FIGURE 21: A PATIENT WITH LEFT FLANK PAIN RADIATING IN THE GROIN AND MICROSCOPIC HAEMATURIA. NO HYDRONEPHROSIS WAS FOUND ON STANDARD ULTRASOUND EXAMINATION. WITH THE DOPPLER ULTRASOUND STUDY, A DIFFERENCE BETWEEN THE (B) LEFT KIDNEY RESISTIVE INDEX (RI) (0.80) AND (A) RIGHT KIDNEY RI (0.59) EMERGED. (C) ON UROGRAPHY, THE LEFT KIDNEY WAS FUNCTIONALLY EXCLUDED (UPPER ARROW) BECAUSE OF A CALCULUS(LOWER ARROW).

The application of proper technique is essential for obtaining accurate results in Doppler USG. The most common reason for obtaining a normal RI in the presence of significant obstruction is a technical error. The use of the correct scale (pulse-repetition frequency) to expand the waveform size to as much of the available display as possible, without aliasing, is crucial.

THEORY OF RI

In a short period immidiately after the obstruction (<2hour) prostaglandin mediated vasodilataion occurs. After this period renal blood flow decreases and renal vascular resistance increases due to complex interactions between several regulatory pathways like renin-angiotensin, kallikrein-kinin and prostaglandin-thromoxane, they causes intense post obstructive renal vasoconstriction. This vasoconstriction was an ideal phenomenon to be detected by the changes demonstrated in RI values.

The elevation in RI values returned to normal after nephrostomy. Accuracy of the Doppler sonographic evaluation of RI in an obstructed kidney was compared with that of the unaffected contralateral kidney, and seen to be elevated in the obstructed kidney where as normal in unobstructed kidney. A difference in RI greater than 0.10 is seen in only true obstruction.

RI is dependent on vascular compliance and resistance , becomes less and less dependant on resistance as compliance decreased.

When complaince is zero RI was completely independent of vascular resistance.

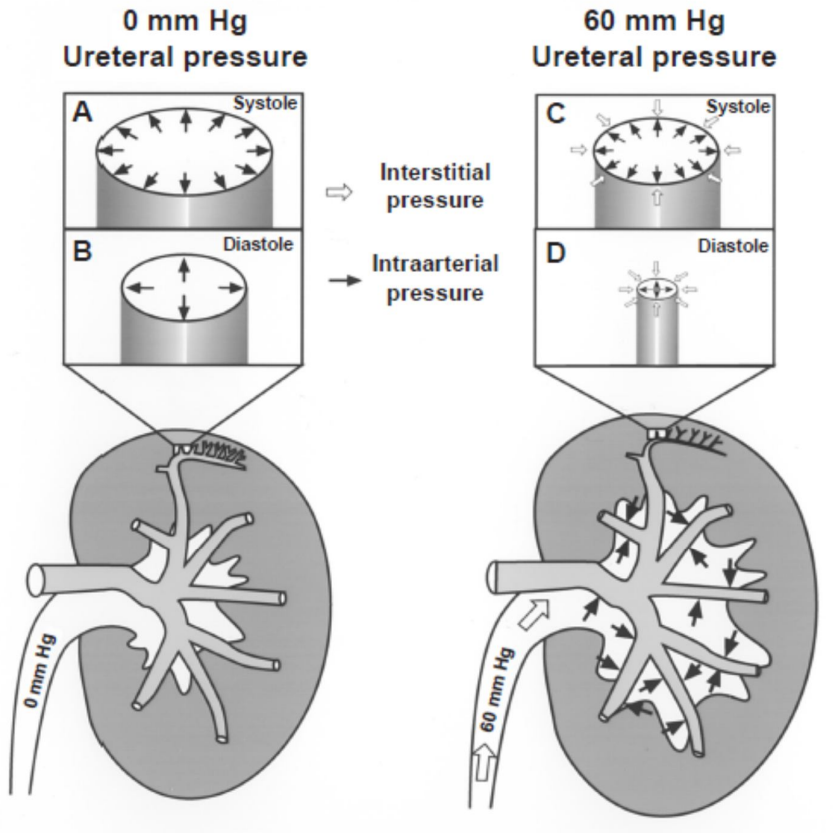


FIGURE 22 : DIAGRAM SHOWING HOW URETERAL PRESSURE WILL AFFECT THE MEAN, SYSTOLIC, AND DIASTOLIC CROSS-SECTIONAL AREAS OF RENAL ARTERIOLES

Area of compliant vessels was determined by the transmural pressure (intraarterial pressure – interstitial pressure).

Interstitial pressure is almost zero in the absence of ureteral pressure (left half of diagram).

During diastole, cross-sectional area of vessel will be relatively large (B), additional distention occurs during systole (A).

High ureteral pressures increases the interstitial pressure (right half of diagram).

Here the arteriole is almost occluded during diastole because transmural pressure is so low (D),there is significant distention occurs during systole (C).

The mean cross-sectional area was markedly smaller with high ureteral pressures (mean conductance of C and D, A and B), relative distention that occurs during systole is greater (conductance of A > B, but C >>D).

These cyclic changes in cross-sectional area were the underlying cause for parallel changes in total renal conductance (flow / pressure).

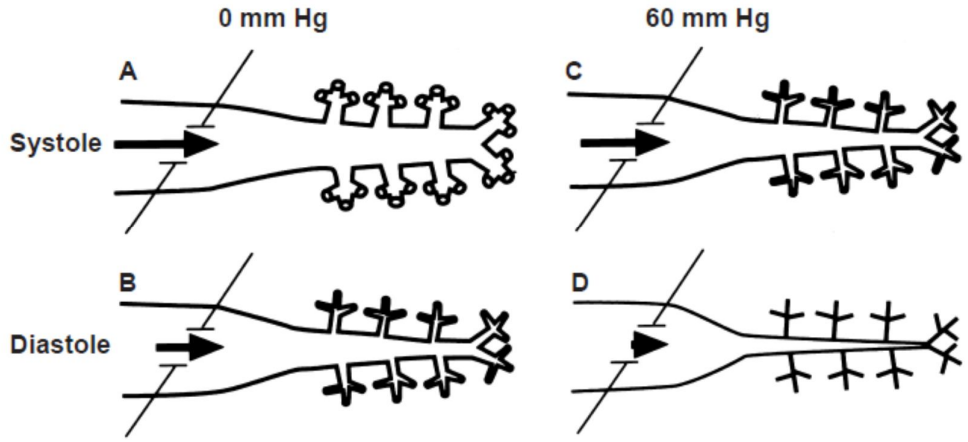


FIGURE 23 : SHOWS HOW URETERAL PRESSURE AFFECTS SYSTOLIC AND DIASTOLIC BLOOD FLOW IN LARGER RENAL ARTERIES TYPICALLY INSONATED DURING CLINICAL RENAL DOPPLER STUDIES.

Each section of figure shows the Doppler gate in conduit (segmental or arcuate) artery, that branches into smaller compliant vessels downstream.

Length of arrow in Doppler gate represents velocity of blood flow. As shown in previous figure changes in ureteral pressure (0 mm Hg in A and B; 60 mm Hg in C and D) significantly affect the arteriolar cross- sectional areas and therefore the total blood flow volume.

The less compliant, larger conduit arteries, changes in blood flow was manifested as cyclic changes in blood velocity.

Thus relative increase in velocity that occurs during systole (measured using resistive index) is greater when ureteral pressure was elevated (velocity in A > B, but C >> D).

REVIEW OF LITERATURE

Sonali S Saboo et al demonstrated that the mean resistivity index in obstructed kidneys were significantly higher than in unobstructed kidneys. The difference in RI between an obstructed and unobstructed kidney (delta RI) ranged from 0.05 - 0.18 and a mean delta RI of 0.08.

Their study showed that most of the patients evaluated between 6-12 hours, within 13-18 hours and 19-24 hours after the onset of symptoms , RI value are similar in all the three groups of people, so there by proving that RI was not a time dependent parameter in their study.

They stated the grey scale ultrasound failed to reveal hydronephrosis in upto 35% of cases in acute obstruction of the kidney. Mild dilatation can be overlooked or can be considered insignificant where as some patients may show no PCS dilatation. PCS dilatation can also be missed if the PCS system is filled with blood clot, calculus tumor or pus.

They also had analyzed the utility of Doppler USG in those patients not having PCS dilatation on gray scale USG, RI values were seen to be higher in all patients with a delta RI of 0.08. There by demonstrated that Doppler USG was useful in diagnosing in those cases where PCS dilation was absent on USG.

In cases of intermittent ureteric obstruction caused by ureteric calculus may lead to failure in visualizing the collecting system with USG.

The direct functional evidence of obstruction usually required scintigraphy, but doppler US techniques is now used to obtain functional information in suspected renal obstruction.

In grey scale ultrasound, false positive rate of diagnosis of obstruction in patients with mild dilatation of PCS can be as high as 26%, and the causes include

(a) Visualisation of a normal PCS system, when there are anatomical variants like extrarenal pelvis, distended bladder, or under conditions of diuresis.

(b) Visualization of a dilated but unobstructed system when there is vesicoureteric reflux, a distended system due to previous obstruction or infection, dilated calyces (in papillary necrosis or reflux nephropathy) or during norm

al pregnancy.

(c) Central renal fluid collections occurring other than PCS, includes the normal vessels, renal artery aneurysm and parapelvic cysts.

By using a statistical value of a threshold RI of ≥ 0.70, their study showed an overall sensitivity of 87.5% and specificity of 90%. At a delta RI of ≥ 0.06 was considered highly sensitive and specific as the sensitivity was 95% and specificity was 100%.

Azmi Haroun stated that 5 % of the urinary calculi that are radiolucent and radio opaque calculi that may lie in a segment of ureter within the bony pelvis caan be confused as phleboliths.

Poor visibility of the ureters was also a major limitation of gray scale renal ultrasonography. The duplex Doppler sonography was seen to improve the clinical utility of ultrasound in patients with urinary obstruction.RI was seen to reflect changes in renal vascular resistance occuring in urinary obstruction. They stated that in complete urinary obstruction RI showed a sensitivity of 64% while that of delta RI were 100%.

Due to an acute urinary obstruction , there occurs an elevation of the pressure in the intrarenal collecting system, causing reduction in renal blood flow, as a result of increased renovascular resistance. Increased RVR leads to decrease in diastolic blood flow velocity in the intrarenal arteries, thereby increasing the RI.

There was no time correlation between the RI values and delta RI values. There study showed a sensitivity of RI to be 64%, after applying delta RI sensitivity rose to 100% and specificity of 89%, suggesting that dRI value is to be measured in acute urinary obstruction.

Study showed the mean RI and dRI in control group of healthy individuals and that of the congtralateral kidneys were 0.59 ± 0.05 and 0.03 ± 0.01, those with obstructed are 0.70±0.06 and 0.09±0.02.

Raza Sayani et al demonstrated that the mean RI for obstructed kidneys were 0.67 that was significantly higher than the mean RI of the contralateral normal kidneys whose mean RI was 0.59. The mean delta RI in patients with acute unilateral renal obstruction was higher than the mean delta RI of the normal kidneys at 0.07 and 0.03.

They found that the positive and negative predictive values of RI in patients with complete urinary obstruction were 79.4% and 82.64%, and that of delta RI were 88.09% and 93.87%. The mean RI values were demonstrated to be 0.67 and 0.59 in acutely obstructed and non obstructed kidneys.The mean delta RI was also seen to be significantly elevated with a value of 0.076.

Damir Miletic et al stated that in obstructed uropathy group mean RI was 0.72 ± 0.04 in the obstructed kidneys and contralateral non obstructed kidneys mean RI was 0.63 ± 0.04, demonstrating that the mean RI in obstructed kidneys were significantly elevated when compared with that of contralateral non obstructed kidneys.

The dRI between the obstructed and unobstructed kidneys were 0.09 ± 0.04. They also measured RIs post relief of the obstruction, showing a decrease in mean RI from 0.72 ± 0.04 to 0.65 ± 0.03 after removal of the obstruction. There was statictically significant differences between RIs and dRIs before and after relief of the obstruction.

According to thier results significant elevation of RI was seen in early obstruction even before dilatation of pelvicalyceal system and ureters have

occurred. After performing an ROC analysis to determine the most appropriate RI they found that an RI of 0.69 or higher or a dRI of 0.06 or higher showed the best sensitivity in diagnosing unilateral acute renal obstruction.

Aneela Azam et al stated that by taking an RI value of >0.70 as a discriminatory level for obstruction, sensitivity of RI was 76.23% and specificity of 88.13%. The PPV of RI is 91.6% and NPV is 68.42% and the diagnostic accuracy of the test was 80%.

Hak Jong Lee et al correlated with symptoms and IVU in 31 patients with unilateral renal obstruction and in 30 control patients. There was no significant correlation between the RI and symptom duration or between RI and pelvicalyceal opacification time on IVU, so they stated that with a cut-off RI of 0.7, sensitivity was found to be 19.3% in unilateral obstruction, but it was increased to 80% in those subjects with acute symptoms and severely delayed pelvicalyceal opacification time, there by suggesting that RI was highly sensitivity in acute and severe obstruction.

Boris Brkjacic et al analysed the utility of color duplex Doppler ultrasonography by performing it in 29 children and adolescents (16 boys, 13 girls) with unilateral dilatation of renal collecting system. 10 patients had acute colic, 7 had PUJ obstruction, 12 having non obstructive hydronephrosis. In detecting obstruction caused by acute colic, RI value ≥0.70 had a sensitivity of 70% and specificity of 92%.

Sensitivity of RI≥0.70 in detecting obstruction due to PUJ obstruction is low (29%). They also demonstrated that various parenchymal diseases and other renal diseases can cause the elevation of renal RI, therefore it was not applied to differentiate obstructive vs non obstructive dilatation. From their data an RIR value of 1.10 or higher was considered the best in distinguishing acute obstruction from non-obstructive hydronephrosis, with a sensitivity of 90% and specificity of 83%. RIR is calculated as the ratio of the higher RI to lower RI.

RIR was a good indicator of obstruction in children with unilateral hydronephrosis caused by PUJ obstruction with a sensitivity of 92% and specificity of 9%.

Joel F Platt et al conducted a duplex Doppler sonography prospective study between the period of 1988 and 1989 in 229 kidneys of 133 patients , in whom 70 kidneys showed dilatation of pelvicalyceal system. In these 70 kidneys 38 kidneys had obstruction.

Obstruction was proved by percutaneous nephrostomy in 24 cases, rest 14 obstructed kidneys obstruction was confirmed by retrograde pyelography, excretory urography, CECT or surgery. 32 of the 70 kidneys were unobstructed, proof of no obstruction was obtained by loopogram in eight, retrograde pyelography in four, and by excretory urography and voiding cystourethrography in one case each.

The mean RI value of 0.77 ± 0.05 in the 38 obstructed kidneys which was significantly different from the RI value 0.63 ± 0.06 in unobstructed kidneys. The results showed a sensitivity of 92% and specificity of 88% for duplex Doppler sonography in the diagnosis of renal obstruction.

Rodgers et al conducted renal examinations and intrarenal arterial Doppler studies in 1990 in 48 patients with normal renal tracts and 20 patients who had presented with acute renal colic due to ureteric calculus. 14 of the patients had urographic evidence of obstruction. They demonstrated , that the obstructed kidneys all had elevated mean RIs higher than their respective non obstructed kidneys.

They have suggested that recent work have documented significant elevation in the intrarenal RI in established renal obstruction and have suggested a discriminatory RI of 0.70 in distinguishing between a dilated obstructed and dilated non obstructed kidneys. They also have stated from their study that the normal range based on 96 kidneys was an average of 6.2 +/- 0.64.

Z Ashraf et al undertook a prospective study over a period of 20 months in 80 obstructed kidneys in 54 patients, to compare the diagnostic accuracy of DDUS and IVP to detect obstruction in thee urinary tract, restricting their study to the kidneys dilated secondary to obstruction.

All patients were clinically evaluated and on suspicion of obstruction those patients was subjected to IVP and DDUS. An RI of >0.70 was considered to be

elevated due to obstruction as was suggested in previous studies by Platt et al. The range of RI in controls were between 0.49 and 0.70 , and a mean of 0.61. IVP had a sensitivity of 85% and specificity of 87.5% in detecting the cause of obstruction, and hydronephrosis was detected with a sensitivity of 92.5% and a false negative rate of 7.5%. B mode USG done after IVP had a sensitivity of 82.5% and specificity of 92.5% in detecting obstruction and a sensitivity of 97.5% , false negative rate of 2.5% in detecting hydronephrosis.

All these patients again underwent DDS, to determine the increase in RI due to increase in resistance to blood flow in both renal and intrarenal arteries. Of the 80 obstructed kidneys 70 had an increase in the RI of > 0.70. In all the 80 kidneys the range was 0.46 to 1.42 with a mean of 0.76. Ten of the obstructed kidneys had an RI <0.70, thus an increased RI had a sensitivity of 87.5% in detecting renal obstruction , specificity of 85% and a false negative of 12.5%.

Kavakli et al investigated the value of renal RI in assessment of renal colic and to determine, its predictive value in renal stone disease. They included a total of 70 patients in there research study. Group I had 43 patients with acute unilateral ureteral obstruction secondary to stone disease. Group II consisted of 7 patients with no stone disease. Group III consisted of 20 healthy patient with two normal kidneys, , comprising the control groups.

All these three groups underwent urinalysis, abdominal plain film radiography, conventional US and CDUS. RI was calculated, compared between the Group

I, Group II and Group III patients. The mean RI was significantly higher in Group I than that for Groups II and III.

The RI value was seen to be raised in Group II patients who had flank pain than those in the control group. RI value was also significantly elevated in those patients with hematuria.

Piazzese et al had conducted a study to determine whether the renal RI could predict HN in patients with renal colic and whether its performance was time dependant. They had included a study population that comprised of 54 patients who was admitted to ED with unilateral renal colic.

Patients were divided into two groups: group A who had presented with signs of dilatation & group B those who did not have signs of dilatation . Each patient underwent routine examinations, abdominal ultrasound, renal CDUS with RI measurement.

The two imaging studies were repeated at 6,12,18,24,36 and 48 hours. The patients also underwent noncontrast urinary tract CT 48-60 hours after admission. A mean renal RI of >0.70 for the symptomatic kidney was considered indicative of obstruction.

The collected data were subjected to statistical analysis for identifying

(a) Possible differences between group A and B

(b) Relation between mRI and time of onset of HN.

(c) Correlation between mRI & the level of , degree and duration of ureteral obstruction.

(d) Sensitivity, specificity, PPV, NPV, accuracy & efficiancy of the method.

They stated that in diagnosing HN ultrasonography displayed excellent sensitivity, specificity, accuracy, PPV & NPV although its performance declined in identifying obstructing and non-obstructing calculosis in groups A &B with a sensitivity of 72.4%, specificity of 64%, accuracy of 68.5%, PPV of 70% and NPV 66.7%. CDUS showed a significant performance in diagnosing the onset of HN with a sensitivity of 100%, specificity of 84%, accuracy of 92.6%,PPV of 79%, NPV 100% and efficacy of 84%.

Group A showed an elevated mRI before the onset of HN(100% of cases) in a time dependant manner. The interval between RI and detection of HN were 6 hours in 19/29 cases (65.5%), 12 hours in8/29 cases (27.6%) & 18 hours in 2/29 cases (6.9 %). This study also demonstrates that there is no significant correlation between mRI values and the level of ureteral obstruction, dilatation, size of calculus and duration of pain. The elevated RI promptly returned to normal once the obstruction was removed.

Hina et al conducted a cross sectional study for a period of 6 months to determine the accuracy of RI in the detection of renal calculi confirmed by IVP. All patients i.e., 170 patients were subjected to DUS to detect renal calculi on

the basis of RI >0.70, after that IVP is done in all patients to confirm the presence or absence of renal calculi. RI was found to be >0.70 in 87% i.e., 148 patients , overall sensitivity of RI was demonstrated to be 87%. Their study confirmed the findings of others that RI can predict the onset of acute obstruction with higher sensitivity, specificity, accuracy and diagnostic efficacy than gray scale US. This study found no significant correlation between the RI and the level of ureteral obstruction.

M.L Ravindernath et al demonstrated in their study that was conducted with the aim to evaluate the use of mean RI as a predictor of hydronephrosis secondary to obstruction in patients admitted with acute renal colic. Sttudy included 84 patients between the ages 15-50 years who were admitted to the EMD with acute unilateral renal colic.

At the time of admission none of the patients had a positive mean resistive index nor hydronephrosis. Eventually 41 cases developed hydronephrosis, and all of them had elevated RI, that developed at different times. After 6 hours of admission no cases demonstrated hydronephrosis, while after 18 hours most of the cases developed hydronephrosis.

DUS demonstrated that after 6 hours of admission 9 had raised RI, after 12 hours 18 patients, after 24 hours 2 patients, after 36 hours 1 patient had elevated RI.

48 hours after the admission no patients had an elevated RI. Therefore this study proves that elevated mean RI has a specificity of 90% and sensitivity of 100%.

Pietro Pepe et al evaluated the addition of a renal color Doppler ultrasonography in the setting of acute renal colic. They evaluated the following parameters like US of both kidneyss, ureters and bladder, RI of renal arteries of both kidneys in three different parennchymal areas, ureteric jets in response to hydration. A renal RI > 0.70 and/ or a difference of 10% between the kidneys were considered as diagnostic of obstructive uropathy.

They had evaluated a total of 100 patients with age ranging between 16-75 years and the study included only those patients who had monolateral renal colic at admission. They performed CDU of the urinary tract within 24 hours of admission. They demonstrated urinary stone as the cause of obstruction in 90 out of the 100 patients. Median RI in obstructed kidneys were 0.73 and in unobstructed kidneys were 0.62 respectively.

Their study proved that in diagnosing an obstruction of the urinary tract conventional US had a high sensitivity of about 98% but low specificity due to presence of 10 - 26% false positive. They also demonstrated in there study that an elevation in RI reflects a significant obstruction, thus having better diagnostic accuracy than conventional US with a sensitivity of 98.9% and specificity of 90.9%.

I.N. Apoku et al stated that there study which had 20 patients with unilateral obstruction, demonstrated an RI value of >0.7 in obstructed kidneys with a sensitivity of 86.7% and specificity of 90%.

OBSERVATION AND RESULTS

In this study of 54 patients who had presented to the EMD with acute unilateral renal colicky pain within 24 hours of onset of symptoms were evaluated with B mode US, CDUS and Duplex Doppler US .

The kidney on the side of obstruction was treated as the case kidney and the contralateral normal (unobstructed) kidney served as the control. Presence or absence of PCS dilatation was assessed in each kidney on the grey-scale images. At least two Doppler spectra were obtained from interlobar arteries along the border of the medullary pyramids and their mean was taken. The Doppler waveforms were made using the lowest pulse repetition frequency possible without aliasing.

TABLE: 2: SEX DISTRIBUTION IN THE STUDY

GENDER	NO OF PATIENTS	PERCENTAGE (%)
Males	**34**	**64**
Females	**20**	**36**

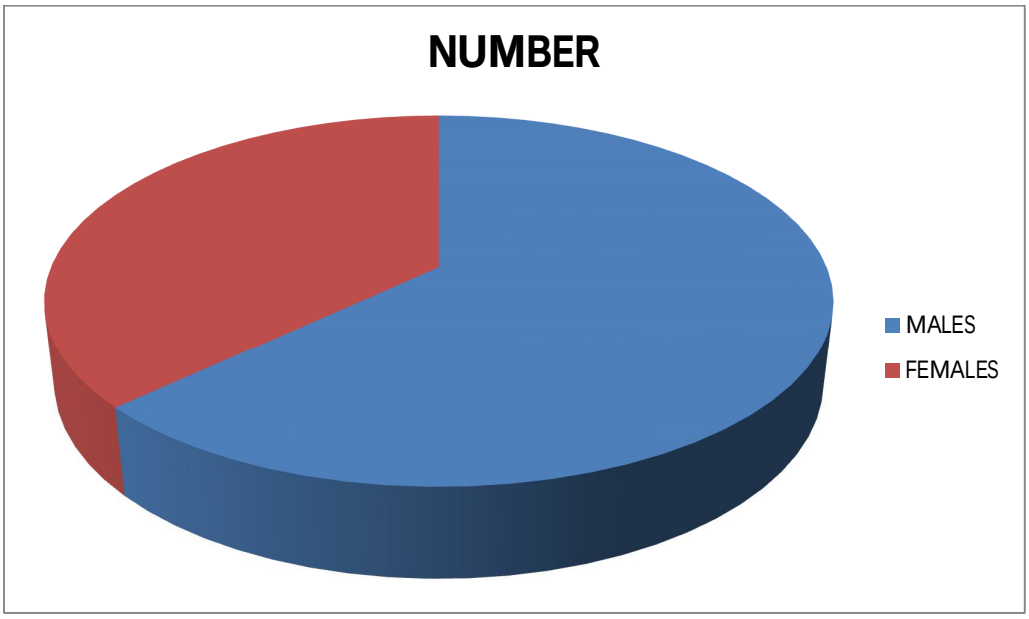


FIGURE 24 : SEX DISTRIBUTION IN THE STUDY

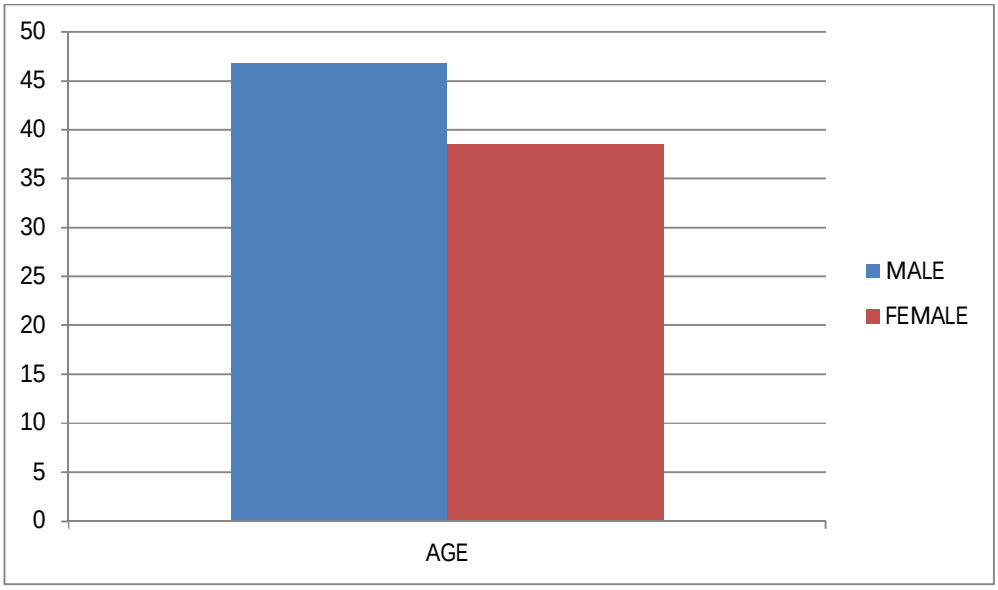
1. In this study population of 54 cases, 34 cases were males and rest 20 females.

TABLE 3 : MODALITY IN DIAGNOSING THE OBSTRUCTION

MODALITY	FREQUENCY	PERCENTAGE
CT	**44**	**81%**
USG	**10**	**19%**
TOTAL	**54**	**100%**

1) Cause of obstruction was confirmed by ultrasound in 10 patients and by CT in 44 patients, and all the patients underwent Duplex Doppler ultrasound to determine the RI.

FIGURE 25 : MEAN AGE OF THE PATIENTS



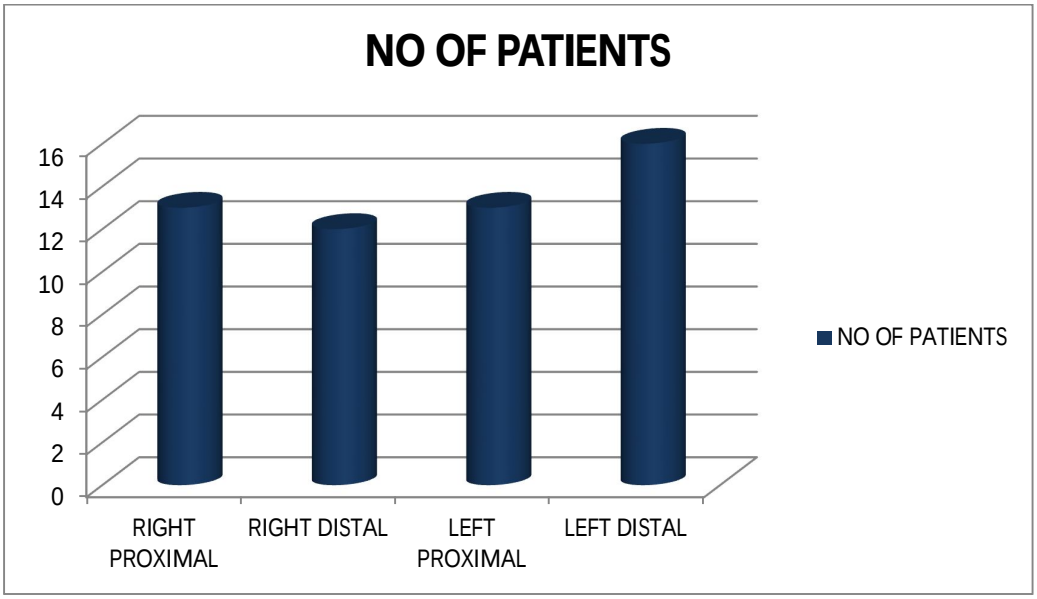
1) Mean age of the male patients were 46.8 years and 38.5 years in female patients.

2) The age range of male patients were 19-79 years and females 18-61 years.

TABLE 4: SITE OF OBSTRUCTION

SITE OF CALCULUS	FREQUENCY
RIGHT PROXIMAL	**13**
RIGHT DISTAL	**12**
LEFT PROXIMAL	**13**
LEFT DISTAL	**16**

FIGURE 26: SITE OF OBSTRUCTION

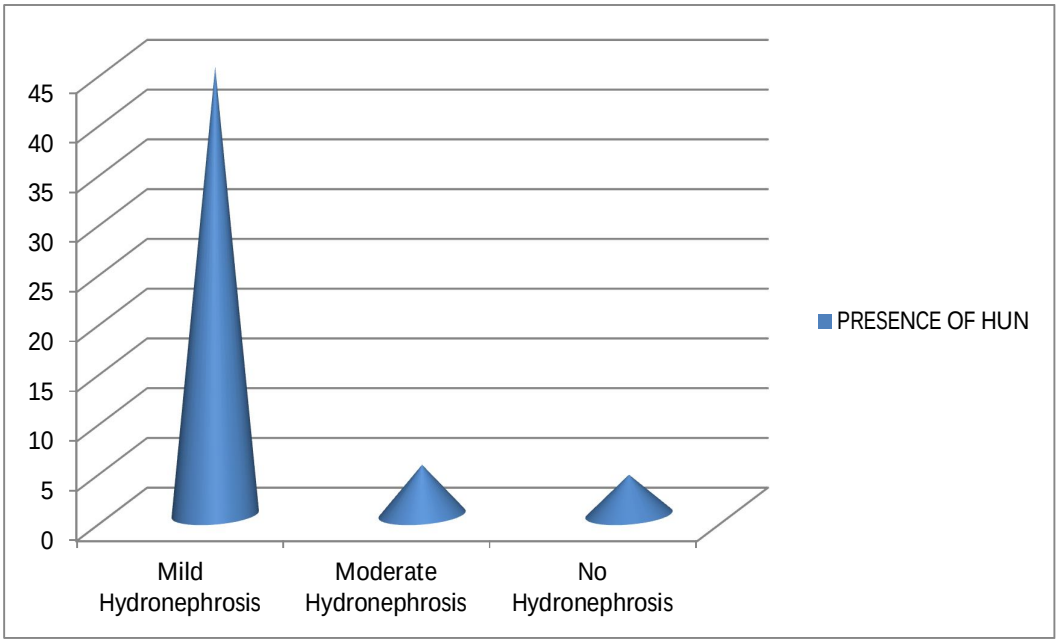


1) Out of 54 patients 26 cases had proximal obstruction while 28 had distal obstruction, The mean RI of the obstructed kidneys was higher in distal obstruction (0.79 ± 0.004), than in the kidneys with proximal obstruction (0.76 ± 0.006).

TABLE 5 : GREY SCALE FINDING IN OBSTRUCTED KIDNEY

OBSTRUCTED KIDNEY	FREQUENCY
MILD HYDRONEPHROSIS	45
MODERATE HYDRONEPHROSIS	5
NO HYDRONEPHROSIS	4

FIGURE 27: FREQUENCY OF PATIENTS WITH HYDRONEPHROSIS



1) Out of the total 54 patients 45 patients had mild hydronephrosis, 5 patients with moderate hydronephrois and 4 patients did not have any dilatation of the collecting system despite of obstruction.

TABLE 6: NUMBER OF PATIENTS WITH ELEVATED SERUM UREA AND CREATININE

NO. OF PATIENTS	SERUM UREA & CREATININE
27	**INCREASED**
27	**NORMAL**

1) Out of the 54 patients who were included in this study, 27 patients had elevated renal function, but did not have history of chronic renal impairment.

FIGURE 28 : RESISTIVITY INDICES IN OBSTRUCTED & UNOBSTRUCTED KIDNEYS

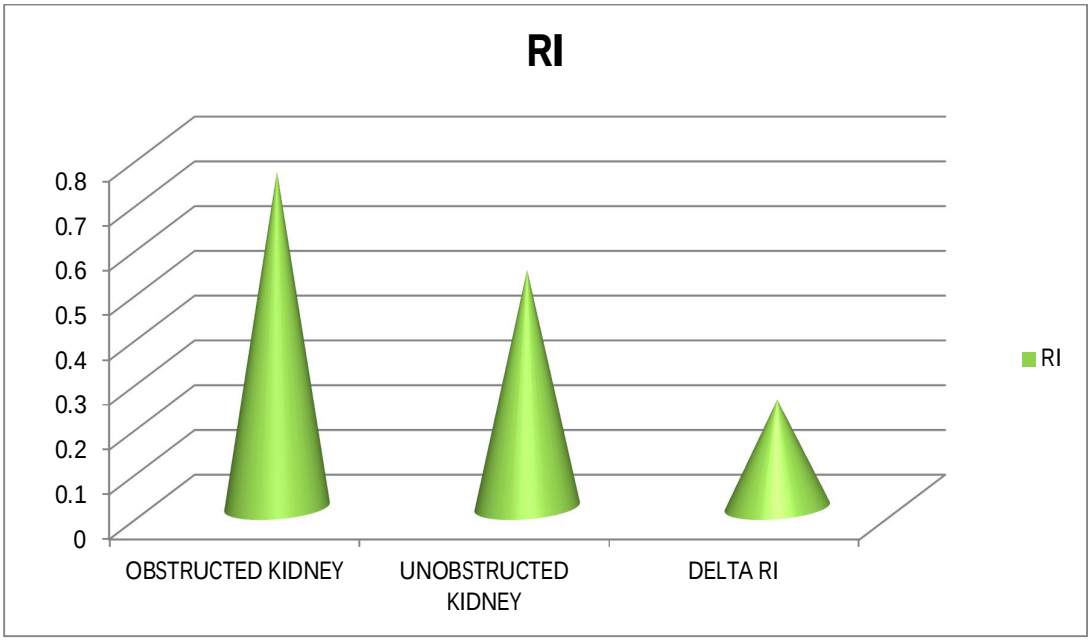
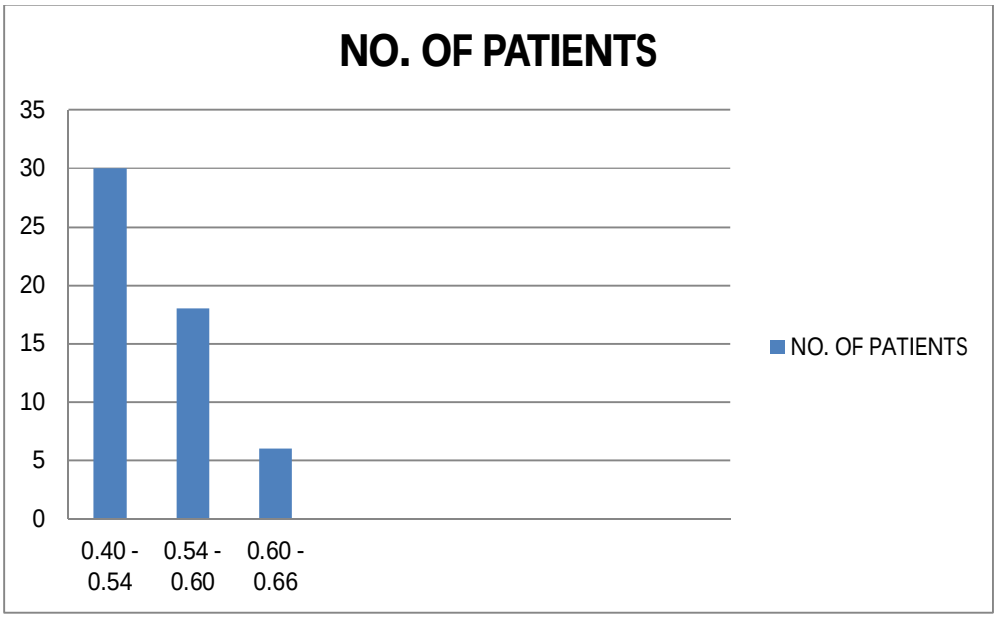


TABLE 7: RESISTIVITY INDICES IN OBSTRUCTED AND UNOBSTRUCTED KIDNEYS

Kidney	RI
Obstructed	0.75 ± 0.08
Unobstructed	0.56 ± 0.09
Delta RI	0.24 ± 0.08

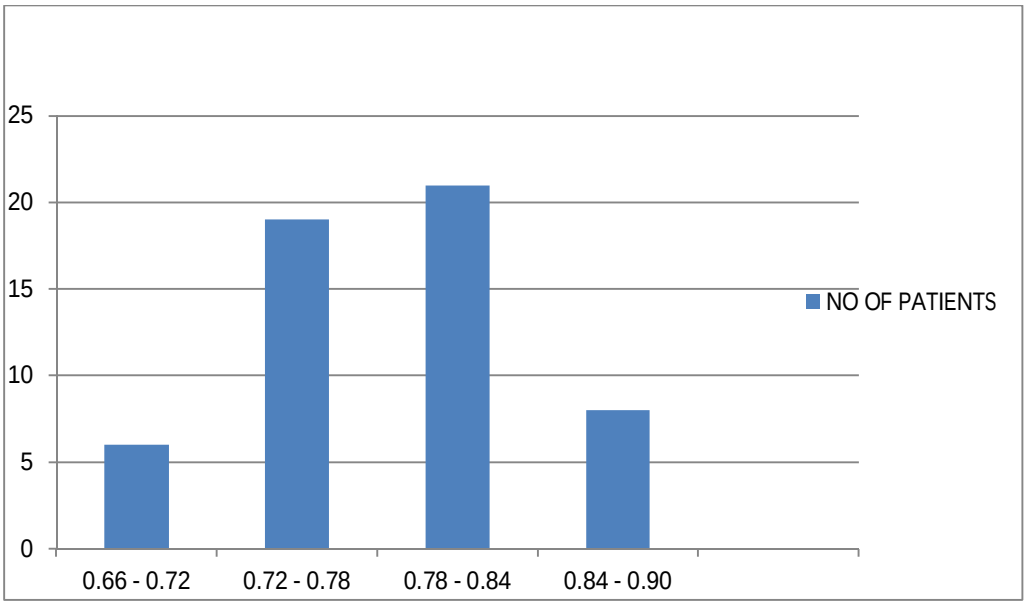
1) In our study of 54 patients, we found that the RI in obstructed kidneys was significantly higher than in the unobstructed kidneys (0.75 Vs 0.56 ; $p < 0.001$). The RI was higher in obstructed kidneys in all cases.
2) The difference between the obstructed and unobstructed kidneys (delta RI) ranged from 0.09 to 0.38 with a mean delta RI of 0.24 .

FIGURE 29: RANGES OF RI VALUES IN THE NORMAL KIDNEYS



1) The mean RI values in unobstructed kidneys ranged from 0.4 – 0.6

FIGURE 30: RANGES OF RI VALUES IN THE OBSTRUCTED KIDNEYS



1) The mean RI values in obstructed kidneys ranged from 0.68 – 0.84

TABLE 8 :GROUP STATISTICS

	FREQUENCY	MEAN RI VALUES	STANDARD DEVIATION	P VALUE
OBSTRUCTED KIDNEY	**54**	**0.75**	**0.08**	**.000***
UNOBSTRUCTED KIDNEY	**54**	**0.56**	**0.09**	

***- statistically significant (P<0.05)**

1) The mean RI value in the obstructed kidney was 0.75 and in unobstructed kidney was 0.56.

TABLE 9: GROUP STATISTICS

	MEAN RI VALUE		P VALUE
	<0.70	**>0.70**	
OBSTRUCTED KIDNEYS	**5 (9.3%)**	**49 (90.7%)**	**0.000***
UNOBSTRUCTED KIDNEYS	**48 (88.9%)**	**6 (11.1%)**	

***-statistically significant (P<0.05)**

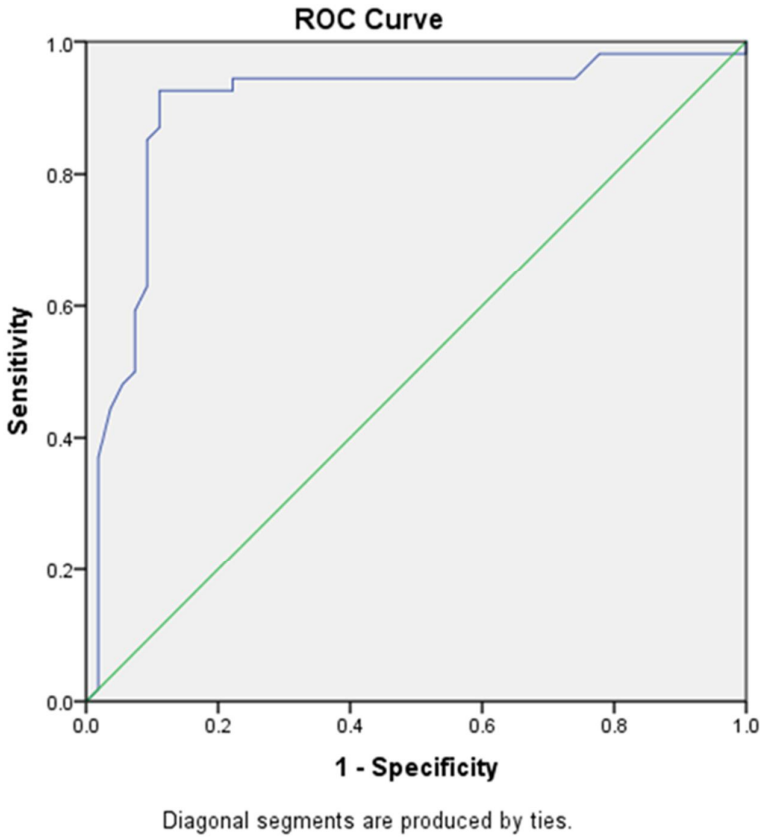


FIGURE 31: ROC CURVE

TABLE 10: SENSITIVITY & SPECIFICITY

	SENSITIVITY (%)	SPECIFICITY (%)
RI > 0.70 IN ACUTE RENAL OBSTRUCTION	**85%**	**93%**

1) By using a cut off RI value of >0.70 in acute unilateral renal obstruction sensitivity obtained in my study was 85% and a specificity of 93%.

RESULTS

In our prospective study of 54 patients who had come to the EMD with history of acute renal colicky pain in less than 24 hours were evaluated initially with B mode US, Color Doppler US and Duplex Doppler US. All the patients were confirmed to have acute obstruction secondary to calculus. 45 patients had mild hydronephrosis, 5 patients had moderate hydronephrosis and rest 4 had no hydronephrosis.

Our study included 20 females and 34 males, age of the females ranging from 18- 61 years and that of males ranging from 19-79 years.

The most common clinical presentation of acute renal obstruction were vomiting, severe lower abdominal pain, flank pain, loin pain radiating to the groin, burning micturition and hematuria.

All the 54 cases had features of unilateral obstruction due to calculus either in the renal pelvis, proximal, mid or distal ureter or in the vesicoureteric junction which had been diagnosed either by US or CT KUB.

44 cases who had obstruction that was not visualized in US, CT KUB was confirmatory. Rest of the 10 cases calculus causing obstruction was demonstrated by US. Doppler Sonography was performed in all cases .

I. Demographic Profile

Of 54 patients, 34 were men and 20 women. The mean age (in years) was 46.85 in men, (19 - 79) was higher compared to 38.5 (18-61) in women. This difference was statistically not significant.

II. Resistivity Indices

The mean Resistivity Index (RI) in obstructed kidneys was significantly higher than in unobstructed kidneys (0.75 Vs 0.56; $p < 0.001$). RI was higher in obstructed kidneys in all the cases. The difference in RI between obstructed and unobstructed kidneys (delta RI) ranged from 0.09 to 0.38 with a mean delta RI of 0.24.

Our study had a sensitivity of 85% and specificity of 93 % by using a discriminatory RI value of 0.70 for acute renal obstruction detected by Doppler US.

III. Factors Affecting Resistivity Indices

1. **Duration of symptoms**:

All 54 patients were evaluated within 24 hrs after the onset of symptoms . Patients with renal colicky pain of more than 24 hours were not included in this study.

2. **Site of obstruction**

The site of obstruction was proximal in 26 (48%) cases and distal in 28 (51%) cases. The mean RI of the obstructed kidneys was higher in distal obstruction (0.79 ± 0.004) than in the kidneys with proximal obstruction (0.76 ± 0.006).

3. **PCS dilatation on greyscale ultrasound**

On gray scale ultrasound, PCS dilatation was observed in 50 (92.5 %) patients while it was absent in 4 (7.4 %). RI values were almost similar in both groups, Those kidneys with dilated pelvicalyceal system RI value was 0.77 ± 0.003, in non-dilated kidneys RI value was 0.75 ± 0.001. 4 of the patients who did not have dilatation of pelvicalyceal system even in the presence of obstruction Doppler sonography was sensitive in diagnosing acute renal obstruction by showing a raised RI value.

IV. Additional parameters

Renal parenchymal thickness :

Parenchymal thickness is a US parameter used in the functional evaluation of the kidney, and a thickness ranging from 15 to 20 mm is considered normal.

All our patients had a renal parenchymal thickness ranging from 15mm -22mm, demonstrating that in acute renal obstruction there is no thinning of the renal parenchyma.

DISCUSSION

Grayscale ultrasound is the investigation of choice in the initial evaluation of acute renal obstruction, as it is easily available, portable and non-ionizing. However, urinary system dilatation seen on conventional gray scale USG has been shown to be sensitive (90%) but not specific (65%-84%) in the diagnosis of renal obstruction.

Traditionally, the evidence of renal obstruction provided by USG has been indirect and dependent on the anatomical criterion of dilatation of the PCS and ureter proximal to the level of obstruction. However, USG fails to reveal hydronephrosis in acute obstruction of the kidney in up to 35% of cases. More direct functional evidence of obstruction has usually required scintigraphy but recently, Doppler US techniques have been used to obtain functional information in suspected renal obstruction.

USG imaging may miss the diagnosis of obstruction in a variety of situations. Mild dilatation may be overlooked or considered clinically insignificant. Some patients with obstructive renal failure may show no PCS dilatation. The reasons for this are unclear; in some patients it may relate to dehydration or to decompression of the pelvicalyceal system by rupture of a calyceal fornix.

PCS dilatation may be missed if the PCS system is filled with blood clot, calculus, tumor or pus. Intermittent ureteric obstruction, particularly caused by ureteric calculi, may also lead to a failure to visualize the collecting system

with USG. On the contrary, in an attempt not to miss the diagnosis of obstruction in patients with only mild PCS dilatation, the false positive rate of diagnosis may be as high as 26%

Causes of a false-positive diagnosis include: (i) Visualization of a normal PCS system, when there are anatomical variants such as extrarenal pelvis, when the bladder is distended or under conditions of diuresis (ii) Visualization of a dilated but unobstructed system when there is vesico-ureteric reflux (VUR), a distensible system after previous obstruction or infection, dilated calyces (e.g. in papillary necrosis or reflux nephropathy) or during normal pregnancy. (iii) Central renal fluid collections other than the PCS, including normal vessels renal artery aneurysm and peripelvic cysts.

Acute unilateral ureteric obstruction results in a complex sequence of changes in renal blood flow and ureteric pressure. In the first two hours, the renal blood flow increases, because of afferent arteriole vasodilatation and the ureteric pressure increases. From two to six hours after obstruction, the renal blood flow decreases, secondary to vasoconstriction of the efferent arterioles and the ureteric pressure remains elevated. Subsequently, at six to eighteen hours, the renal blood flow remains reduced, because of vasoconstriction of the afferent arterioles and the ureteric pressure decreases.

The role of renal Doppler USG in the evaluation of acute renal obstruction has been vigorously debated . **Rodgers *et al*** found an elevated RI in acutely obstructed kidneys, especially when compared with the RI in normal contralateral kidneys and with a control group of healthy subjects. Similar results were obtained by **Platt *et al*** in 23 patients with acute unilateral ureteric obstruction. However, others have reported that Doppler ultrasound is highly insensitive for detecting acute renal colic. **Tublin *et al*** correlated the results of Doppler USG with those of urography in 32 patients presenting with symptoms of renal colic. When the published discriminatory thresholds for obstruction (mean RI $\geq$ 0.70 and ΔRI $\geq$ 0.10) were applied, the sensitivity and specificity of Doppler USG were only 44% and 82%, respectively.

In our study of 54 patients, we found that the RI inobstructed kidneys was significantly higher than in the unobstructed kidneys (0.75 Vs. 0.56; $p < 0.001$). The RI was higher in obstructed kidneys in all cases. The difference between the obstructed and unobstructed kidneys (deltaRI) ranged from 0.09 to 0.38 with a mean delta RI of 0.24.

Sonali Saboo et al stated in their study that delta RI $\geq$0.006 was highly sensitivity and specific and with a higher delta RI specificity remained the same whereas the sensitivity fell rapidly.

Our results correlate well with many studies reported earlier by Badr K et al, Shokeir AA, Miletic D.

We also studied the effect of the level of obstruction on RI values. In our study the level of ureteric obstruction (proximal v/s distal), distal obstruction had more RI value (0.79) compared to proximal obstruction (0.76) and the values were not statistically significant. However, the study done by de **Toledo *et al*** showed that patients with proximal ureteric obstruction have RIs higher than those with distal obstruction .

We also analyzed the utility of Doppler USG in patients not having dilatation of the PCS on ultrasound. patients who did not have PCS dilatationon USG were later confirmed to have obstruction on CT. RI values were higher in all these patients (0.71) with a Delta RI of 0.24. Discriminatory RI $\geq$ 0.70 were found to be highly sensitive (85%) and specific (93 %) in this study which is higher than the study done by Platt JF *et al*

SUMMARY

- Doppler changes in acute unilateral renal obstruction were studied in 54 patients with contralateral normal kidney as control.
- The age of the patients ranged from 18 – 79 years and out of 54 patients, 34 were men and 20 women.
- The mean Resistivity Index (RI) in obstructed kidneys was significantly higher than in unobstructed kidneys (0.75 vs 0.56; $p < 0.001$).
- The distal obstruction showed more RI value i,e., 0.79 than proximal obstruction RI value 0.76, and it was statistically not significant.
- Doppler changes were positive in 4 patients even when there was no pelvicalyceal system dilatation on ultrasound.
- My study showed a sensitivity of 85% and specificity of 93 % for detecting acute unilateral renal obstruction with a cut off RI of 0.7 .

CONCLUSION

Using the discriminatory threshold value of RI ≥ 0.70, the overall sensitivity of Doppler USG in diagnosing acute renal obstruction was 85% and specificity was 93%. Doppler ultrasound study is useful diagnostic tool in suspected acute unilateral renal obstruction as Renal Doppler recording can demonstrate altered renal perfusion before pelvicalyceal system dilatation thus increasing the accuracy of ultrasound in the investigation of renal disease. Gray-scale sonography therefore should be complemented by Doppler US as an adjunctive test in patients presenting with acute renal colic.

It is very simple, noninvasive and easy to calculate and can be routinely used as a predictor to hydronephrosis in patients with acute renal colic.

Special cases were ionising radiations are undesirable, such as in pregnant patients or patients of paediatric age group, Doppler US can be used to reach conclusion.

This study supplements the existing evidence that renal Doppler recordings can distinguish between acutely obstructed and non-obstructed kidneys by demonstrating changes in renal blood flow.

Imaging plays an important role in diagnosis, treatment planning, and post-treatment follow-up of urolithiasis. As renal calculus disease is becoming more prevalent worldwide, accurate and appropriate imaging will help optimize treatment and minimize complications of nephrolithiasis and their associated

cost. Accurate reporting of stone location, size, density, internal structure are helpful for the referring clinicians and often affects patient management. Given the high propensity of patients with urinary tract calculi to undergo multiple imaging studies, minimizing radiation dose while maintaining acceptable diagnostic accuracy is of paramount importance.

LIMITATIONS

- US is an operator dependent examination and the results are much affected by patient body habitus.
- Increase in RI of intrarenal vessels is also detectable in case of vascular or parenchymal diseases (renal vein thrombosis, acute renal insufficiency, renal artery stenosis, etc.)
- The major problem with the technique has been technical error, either related to the technology used or the experience of the operator.

REPRESENTATIVE CASES

Case 1

54 year old female came with right flank pain and vomiting

Figure 32 (A) B mode US demonstrating calculus of size 4.1 mm in the right distal ureter

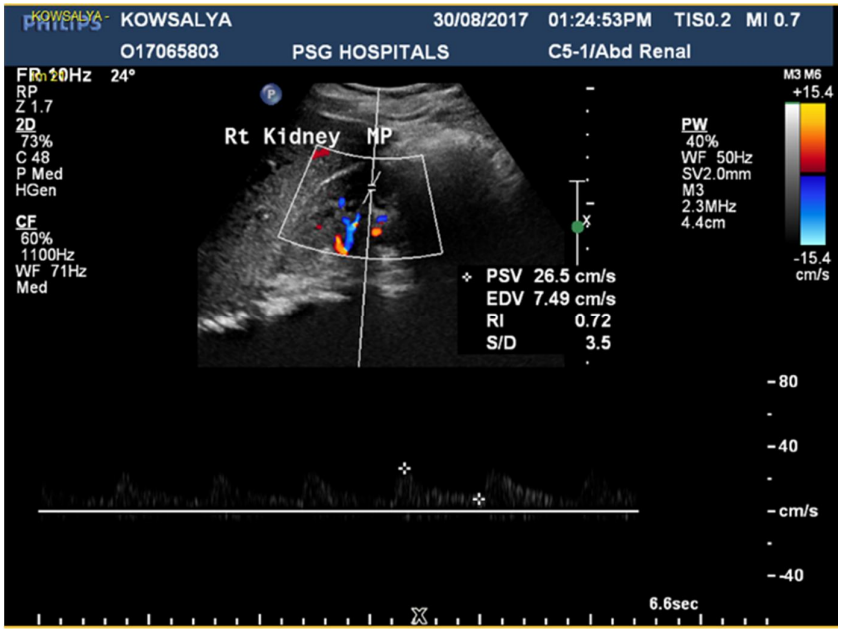


Figure 32 (B) Doppler US demonstrating an RI value of 0.72 in the mid pole right kidney (obstructed).

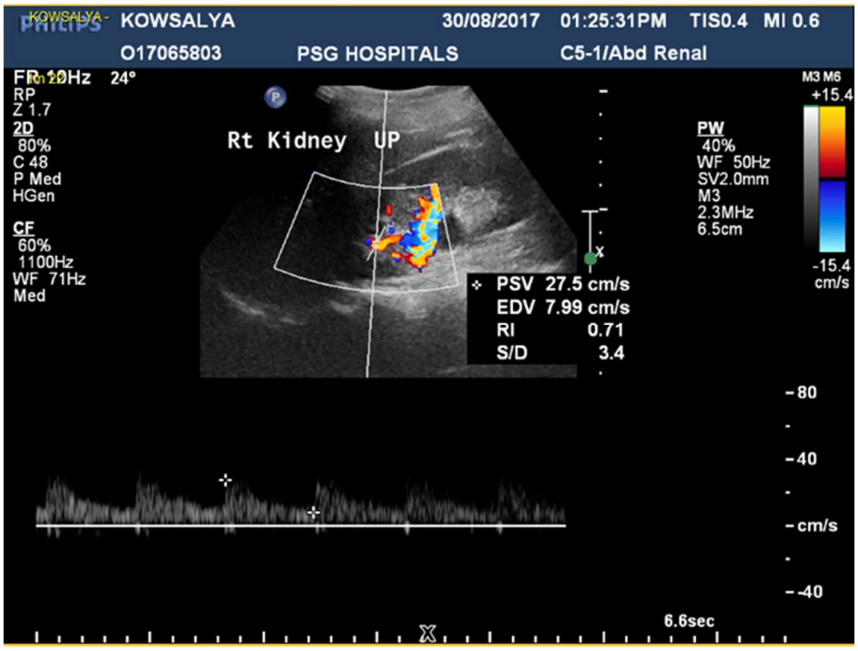


Figure 32 (C) Doppler US demonstrating an RI value of 0.71 in the upper pole of right kidney (obstructed).

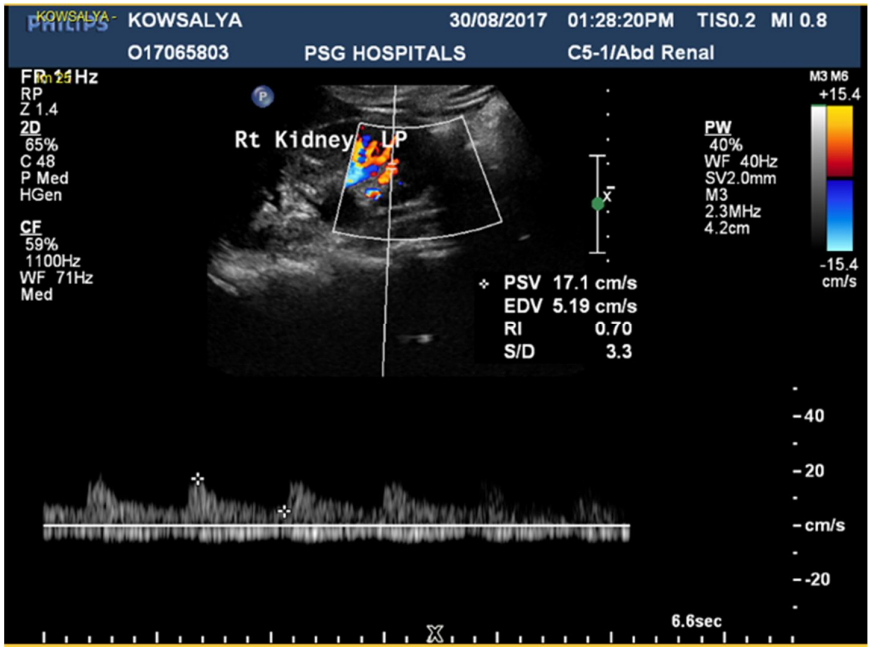


Figure 32 (D) Doppler US demonstrating an RI value of 0.70 in the lower pole of right kidney (obstructed).

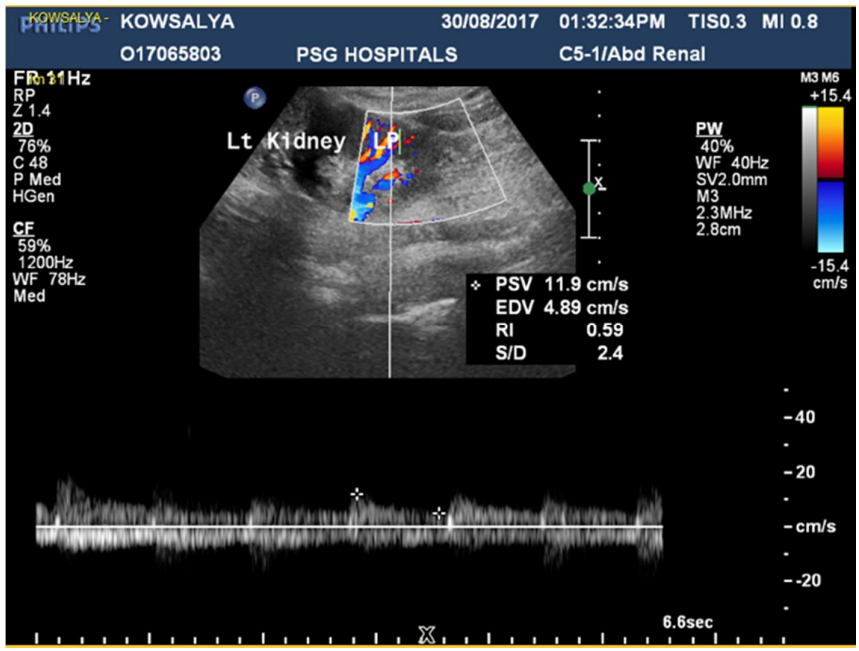


Figure 32 (E) Doppler US demonstrating an RI value of 0.59 in the lower pole of left kidney(unobstructed).

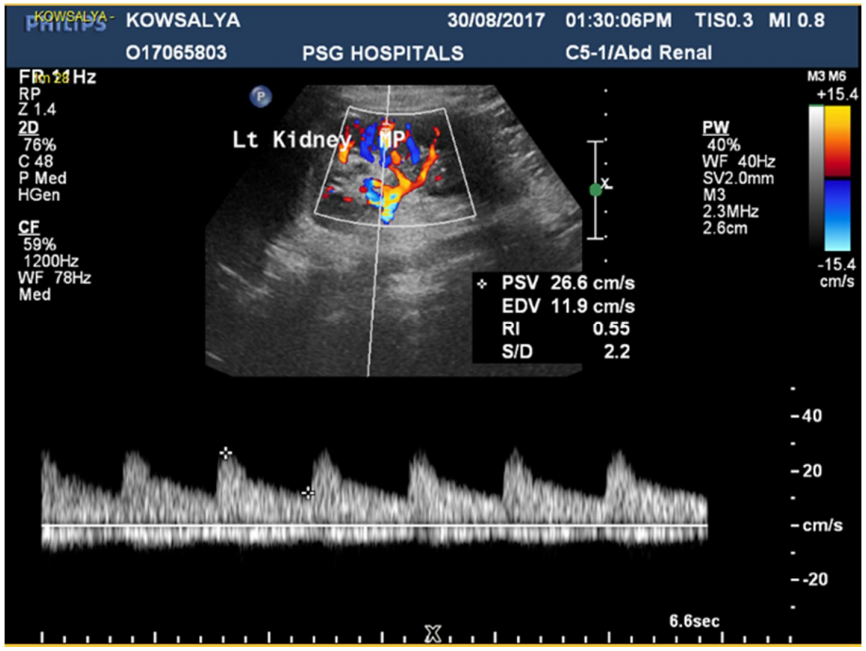


Figure 32 (F) Doppler US demonstrating an RI value of 0.55 in the mid pole of left kidney(unobstructed).

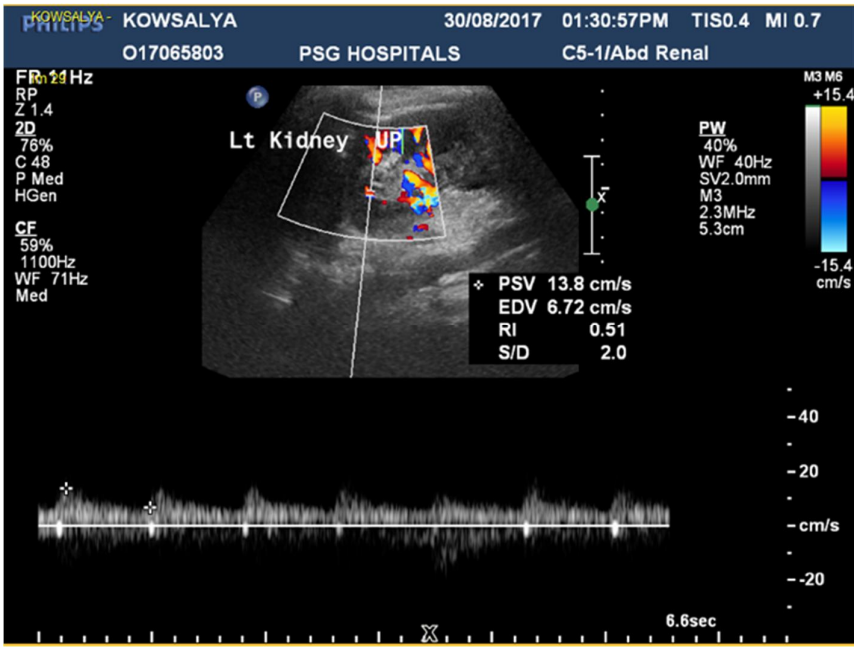


Figure 32 (G) Doppler US demonstrating an RI value of 0.51 in the upper pole of left kidney(unobstructed)

Case 2 54 year old female patient with left flank pain radiating to loin

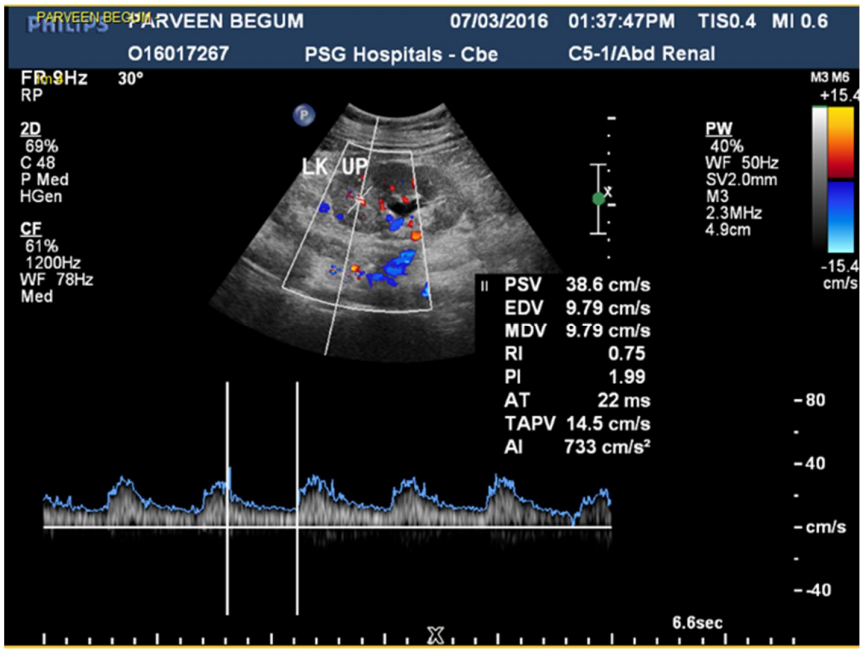


Figure 33 (A) Doppler US demonstrating an RI value of 0.75 in the upper pole of left kidney (obstructed kidney)

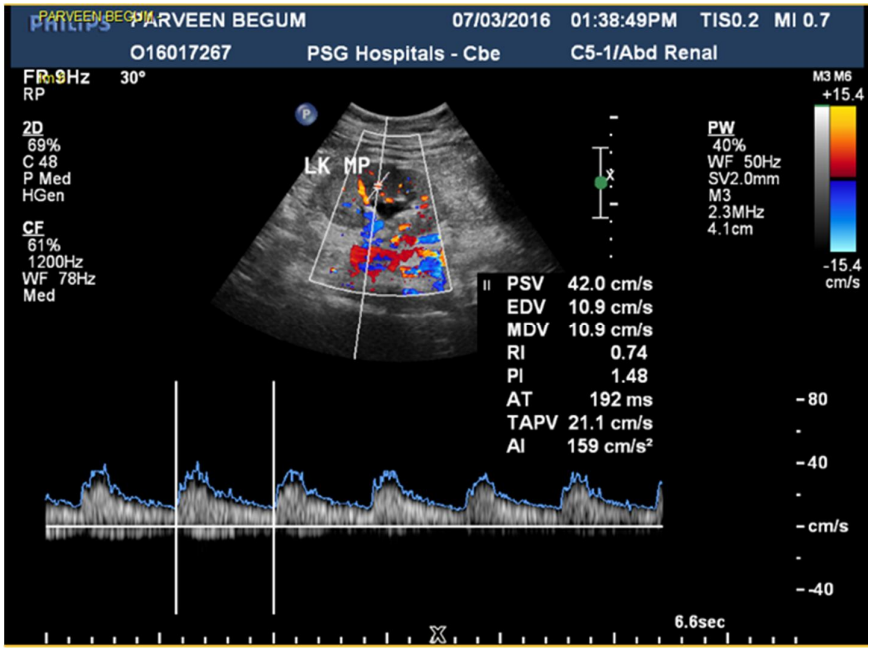


Figure 33 (B) Doppler US demonstrating an RI value of 0.74 in the mid pole of left kidney (obstructed kidney)

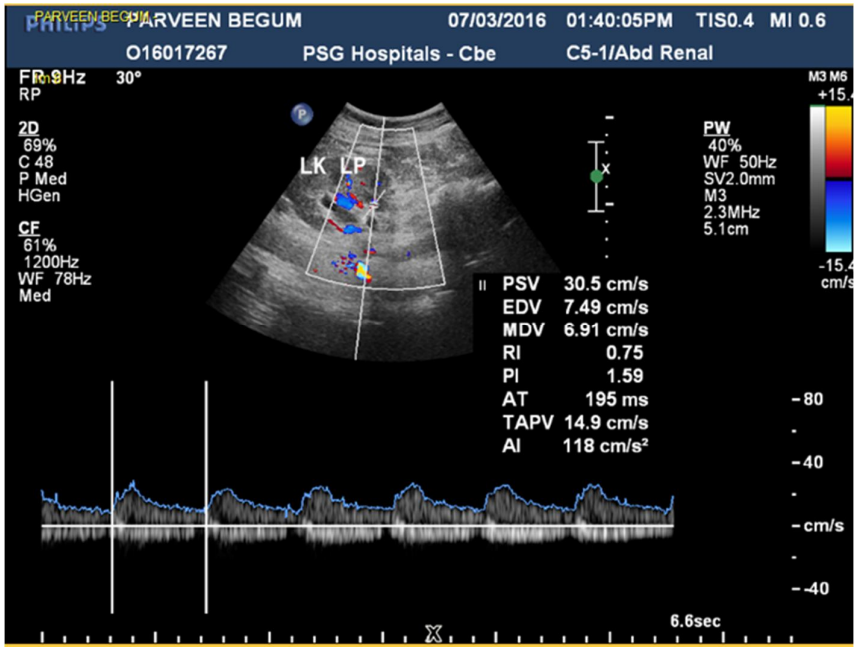


Figure 33 (C) Doppler US demonstrating an RI value of 0.75 in the lower pole of left kidney (obstructed kidney)

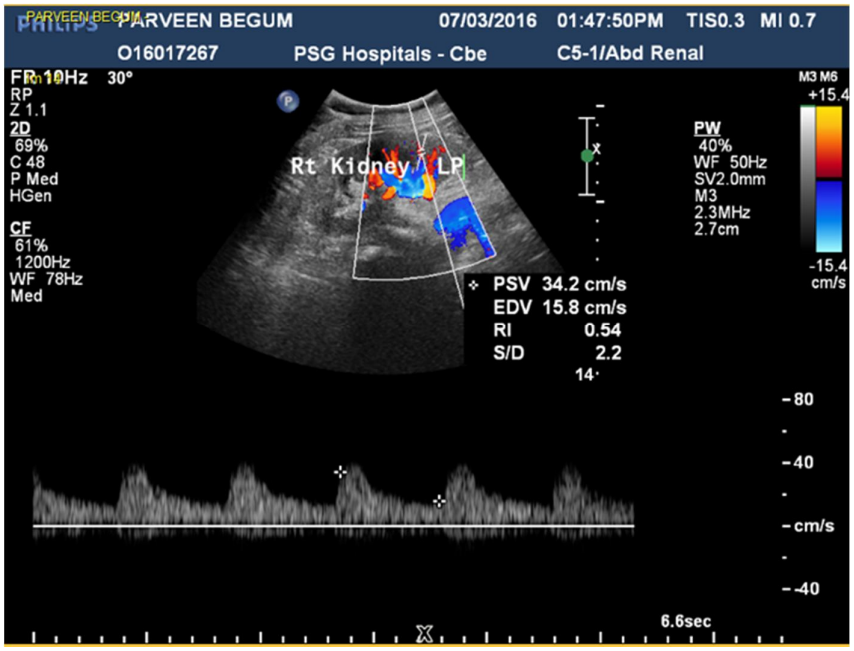


Figure 33 (D) Doppler US demonstrating an RI value of 0.54 in the lower pole of right kidney (unobstructed kidney)

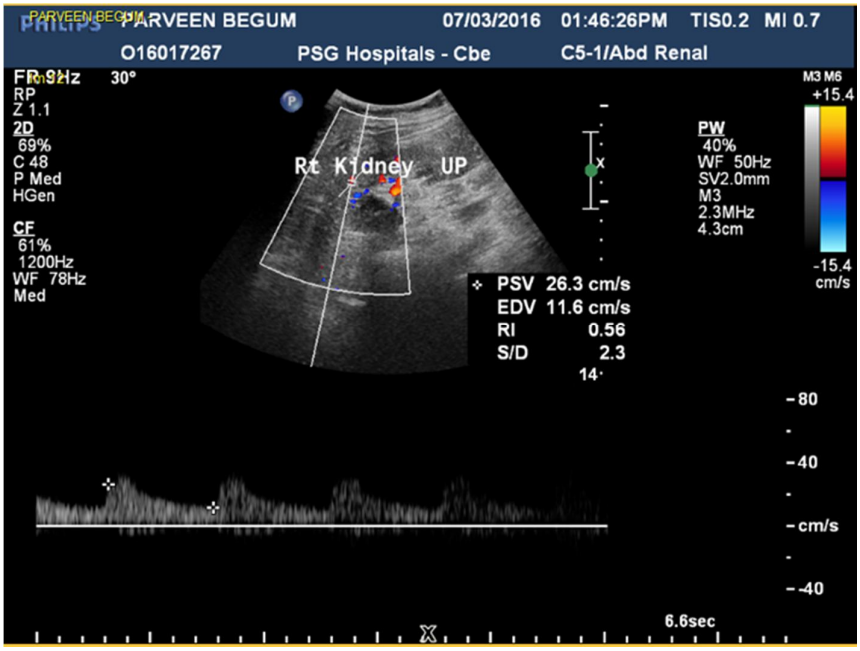


Figure 33 (E) Doppler US demonstrating an RI value of 0.56 in the upper pole of right kidney (unobstructed kidney)

Figure 33 (F) Sagittal plain CT abdomen demonstrating a calculus in the left ureter.

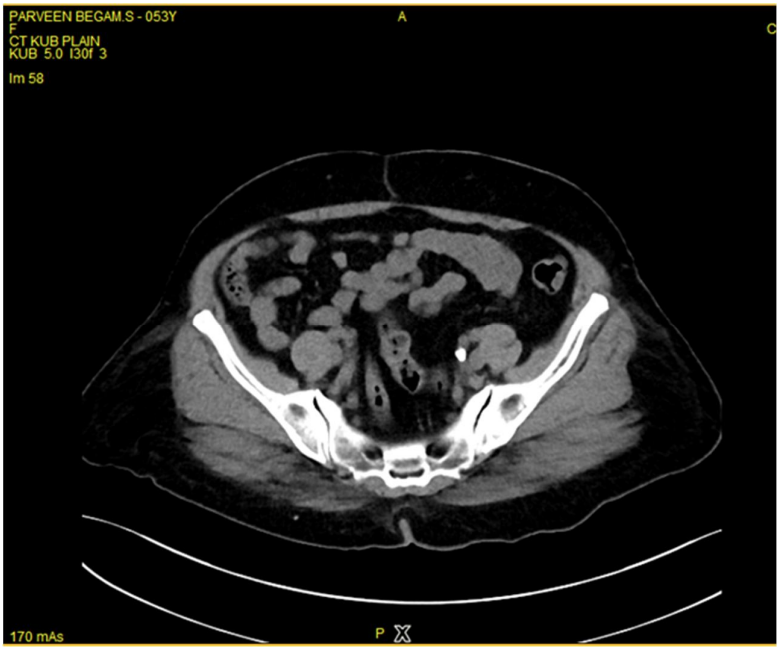


Figure 33 (G) Axial plain CT demonstrating calcus in left distal ureter.

BIBLIOGRAPHY

1) Sonali S Saboo, Sachin S Soni, Suresh H Saboo, Naga Ramesh Chinapuvvula, Sashidhar Kaza. Doppler sonography in acute renal obstruction . IJRIE August 2007 / Vol 17 /188-192 Issue 3.

2) Haroun Azmi. Duplex Doppler sonography in patients with acute renal colic: Prospective study and literature review. Int Urol Nephrol. 2003;35(2):135-40.

3) Sayani R , Ali M, Shazlee K, Hamid RS, Hamid K. Functional evaluation of the urinary tract by duplex Doppler ultrasonography in patients with acute renal colic. Int J Nephrol Renovasc Dis 2012;5:15-21.

4) Miletic D, Fuckar Z, Sustic A, Mozetic V, Smokvina A, Stancic M. Resistance and pulsatility indices in acute renal obstruction, J Clin Ultrasound. 1998 Feb; vol 26(2):79-84.

5) Azam Aneela, Arfan-ul-haq , Beg MA. Role of renal arterial resistive index (RI) in obstructive uropathy. J Pak Med Assoc. 2013 Dec;63(12):1511-5.

6) Lee H J, Kim S H, Jeong Y K , Yeun KM. Doppler sonographic resistive index in obstructed kidneys. J Ultrasound Med. 1996 Sep;15(9):613-8.

7) Brkljacic B, Drinkovic I, Sabljar-Matovinovic, M, Soldo D, Morovic-Vergles J, Vidjak V et al. Doppler sonographic renal resistance index

and resistance index ratio in children and adolescents with unilateral hydronephrosis. Eur Radiol. 2002 Nov;12(11):2747-5.

8) Platt, J. F., Rubin, J. M. , Ellis, J. Distinction between obstructive and nonobstructive pyelocaliectasis with duplex Doppler sonography. AJR Am J Roentgenol. 1989 Nov;153(5):997-1000.

9) Rodgers P. M , Bates J. A , Irving H. C.Intrarenal Doppler ultrasound studies in normal and acutely obstructed kidneys. Br J Radiol. 1992 Mar;65(771):207-12.

10) Kavakli H S, Koktener A, Yilmaz A. Diagnostic value of renal resistive index for the assessment of renal colic. Singapore Med J. 2011 Apr;52(4):271-3.

11) Piazzese E.M , Mazzeo G.I, Galipò S, Fiumara F, Canfora C, Angiò LG. The renal resistive index as a predictor of acute hydronephrosis in patients with renal colic. J Ultrasound. 2012 Oct 14;15(4):239-46.

12) Gul, Hina Habib, Irum Roghani, Inayat Shah. Diagnostic accuracy of renal arterial resistive index (RI) in acute renal colic KJMS. January-June. 2014 Vol. 7, No.1.

13) Ravindernath, M. L and *G.* Mahender Reddy.Mean resistive index as a prognostic tool for hydronephrosis in patients with acute renal colic : a study in a tertiary care. Int J Adv Med. 2017 Apr;4(2):329-333.

14) Ashraf, Z Mansoor, T Ashai, M Ahmad, W Lateef . Duplex Doppler Ultrasonography: An Excellent Initial Investigation In Obstructive Uropathy .The Internet Journal of Surgery. 2008 Volume 20 Number 1.

15) Pepe Pietro, Motta Luigi, Pennisi, Aragona F. Functional evaluation of the urinary tract by color-Doppler ultrasonography (CDU) in 100 patients with renal colic. Eur J Radiol. 2005 Jan;53(1):131-5.

16) Apoku I N, Ayoola , Salako AA, Idowu BM. Ultrasound evaluation of obstructive uropathy and its hemodynamic responses in southwest Nigeria. Int Braz J Urol. 2015 May-Jun;41(3):556-61.

17) Wayne Brisbane, Michael R. Bailey, Mathew D. Sorensen. An overview of kidney stone imaging techniques. Nat Rev Urol. 2016 Nov;13(11):654-662.

18) Kiliç S, Altinok MT, Ipek D, Beytur A, Baydinç YC, Guneş G. Color Doppler sonography examination of partially obstructed kidneys associated with ureteropelvic junction stone before and after percutaneous nephrolithotripsy: Preliminary report . Int J Urol. 2005 May;12(5):429-35.

19) Granata A, Zanoli L, Clementi S, Fatuzzo P, Di Nicolo P, Fiorini F.Resistive intrarenal index: Myth or Reality? Br J Radiol. 2014 Jun;87(1038):20140004.

20) Kambadakone Avinash R, Eisner Brian H, Catalano, Sahani DV. New and evolving concepts in the imaging and management of urolithiasis: urologists' perspective. Radiographics. 2010 May;30(3):603-23.

21) Portis, Andrew J. Sundaram, Chandru P. Diagnosis and initial management of kidney stones. Am Fam Physician. 2001 Apr 1;63(7):1329-38.

22) Parolini C, Noce A, Staffolani E, Giarrizzo GF, Costanzi S, Splendiani G. Renal resistive index and long term outcome in chronic nephropathies. Radiology. 2009 Sep;252(3):888-96.

23) Gurel S, Akata D, Gurel K, Ozmen MN, Akhan O.Correlation between the renal resistive index (RI) and nonenhanced computed tomography in acute renal colic: how reliable is the RI in distinguishing obstruction? J Ultrasound Med. 2006 Sep;25(9):1113-20.

24) Veltri A, Serrallonga M, Santoro B, Zanon E, Gilardi C, Cornaglia A, Galli J Doppler ultrasonography of the intrarenal arteries before and after radiologic treatment in obstructive Uropathy. Radiol Med. 1995 Jul-Aug;90(1-2):70-4.

25) Karadeniz T, Topsakal M, Eksioğlu A, Ariman A, Basak D. Renal hemodynamics in patients with obstructive uropathy evaluated by color Doppler sonography Eur Urol. 1996;29(3):298-30.

26) Soylu Boy, Kayhan A, Karakas HM , Alp T, Verit A. Diffusion-weighted MR imaging and Doppler ultrasonography in the evaluation of renal parenchyma in acute ureteral Obstruction. International journal of clinical and experimental medicine. Int J Clin Exp Med. 2015 Feb 15;8(2):2719-26.

27) Pelaez LI, Juncos LA, Stulak JM, Lerman LO, Romero JC. Invasive evaluation of bilateral renal regional blood flow and tubular dynamics during acute unilateral ureteral obstruction. Nephrol Dial Transplant. 2005 Jan;20(1):83-8.

28) Ucero AC, Benito-Martin A, Izquierdo MC, Sanchez-Niño MD, Sanz AB, Ramos AM, Berzal S. Unilateral ureteral obstruction: beyond Obstruction, Int Urol Nephrol. 2014 Apr;46(4):765-76.

29) Brown, Carlo L Rosen, Wolfe R E. Renal ultrasonography, Emergency Medicine Clinics of North America. ISSN: 0733-8627, Vol: 15, Issue: 4 Page: 877-893.

30) Krumme B, Renal Doppler sonography--update in clinical nephrology. Nephron Clin Pract. 2006;103(2):c24-8.

31) Tublin M E, Bude R O , Platt JF. The resistive index in renal Doppler sonography: Where do we stand? AJR Am J Roentgenol. 2003 Apr;180(4):885-92.

32) Kim S. H, Kim W. H, Choi B. I, Kim CW. Duplex Doppler US in patients with medical renal disease: Resistive index vs serum creatinine level. Clin Radiol. 1992 Feb;45(2):85-7.

33) Opdenakker L, Oyen R, Vervloessem I, Goethuys H, Baert AL, Baert LV et al. Acute obstruction of the renal collecting system: the intrarenal resistive index is a useful yet time dependent parameter for diagnosis. Eur Radiol. 1998;8(8): 1429-32.

34) *V. D'Addario , Donald School Basic Textbook of Ultrasound in Obstetrics &* Gynecology. JP Medical Ltd. *Page 189*

35) Inderbir Singh. Urogenitalsystem. Human embryology.8th ed. New Delhi: MacMillan India Ltd; 2007.p. 237-245.

36) Sadler T.W. Urogenital system. In: Sadler T.W, editor. Medical embryology. 7th ed. Baltimore: Williams and Wilkins; 1995. p. 272-311.

37) Inderbir Singh. Deep structures of the neck. In: Inderbir Singh. B.D. Chaurasia's Human Anatomy regional and Applied, Volume Two Lower limb and Abdomen. New Delhi: CBS publishers; 1993.p. 257.

38) Neil R. Barley, Jeremiah C.Healy, Patricia Collins, David Johnson, Alan Crossman. Urogenital system. In: Susan Standing, editor. Gray's Anatomy. 40th ed. Edinburgh, London: Churchill-Livingstone; 2008.p.1225 -1230

39) Moore, K.L. The abdomen. In: Moore, K.L. editor. Clinically oriented anatomy. 4th ed. Baltimore: Lippincott Williams and Wilkins; 1999.p.280- 284.

40) Meschan I. The Urinary Tract. In: Meschan I, editor. An Atlas of anatomy basic to radiology.Philadelphia: W.B.Saunders Co; 1975.p.998-1055.

41) Arthur C. Guyton. Function of kidney. In Arthur C. Guyton, editor.Textbook of Medical Physiology.10th edition. W.B Saunder`s company; 2000.p.279 – 280.

42) Arthur C. Guyton. Formation of urine by the kidney: Glomerular Filtration, Tubular Function and Plasma Clearance. In: Arthur C. Guyton, editor. Textbook of Medical Physiology.10th edition. W.B Saunder`s company; 2000.p.282 – 284.

43) Woo J and Henry JB. Metabolic intermediates and inorganic ions. In: Henry JB, editor. Clinical Diagnosis and Management by Laboratory Methods. 19th ed. Philadelphia: W.B Saunders company; 1996. p.162-165.

44) Deb. A. C. Fundamentals of Biochemistry. Calcutta: New Central Book Agency (P) Ltd; 1992.p. 293 - 318.

45) Hankins DA, Babb AL, Uvelli DA, Scribner BH. Creatinine degradation I: the kinetics of creatinine removal in patients with chronic kidney disease. Int J Artif Organs 1981 Jan; 4(1):35-39.

46) Vasudevan D. M., Sreekumari S. Textbook of Biochemistry for Medical students, 6th edition. New Delhi: Jaypee brothers medical publishers (P) Ltd; 2011.p. 320.

47) Anderson JK, Kabalin JN, Cadeddu JA. Surgical anatomy of retroperitoneum, adrenals, kidneys, and ureters. In: Wein AJ, ed. Campbell-Walsh Urology. Vol 1. 9th ed. Philadelphia, Pa: Saunders Elsevier; 2007:3-37

48) Brooks JD. Anatomy of the lower urinary tract and male genitalia. In: Wein AJ, ed. Campbell-Walsh Urology. Vol 1. 9th ed. Philadelphia, Pa: Saunders Elsevier; 2007:38-77.

49) Tanagho EA. Anatomy of the genitourinary tract. In: Tanagho EA, McAninch JW, eds. Smith's General Urology. 17th ed. New York, NY: McGraw-Hill; 2008:Ch. 1.

50) Worcester EM, Coe FL. Nephrolithiasis. Prim Care. Jun 2008;35(2):369-91.

51) Evan AP, Coe FL, Lingeman JE, Shao Y, Sommer AJ, Bledsoe SB, et al. Mechanism of formation of human calcium oxalate renal stones on Randall's plaque. Anat Rec (Hoboken). Oct 2007;290(10):1315-23.

52) Lindqvist K, Hellström M, Holmberg G, Peeker R, Grenabo L. Immediate versus deferred radiological investigation after acute renal colic: a prospective randomized study. Scand J Urol Nephrol. 2006;40(2):119-24.

53) Jindal G, Ramchandani P. Acute flank pain secondary to urolithiasis: radiologic evaluation and alternate diagnoses. Radiol Clin North Am. May 2007;45(3):395-410.

54) Jindal G, Ramchandani P. Acute flank pain secondary to urolithiasis: radiologic evaluation and alternate diagnoses. Radiol Clin North Am. May 2007;45(3):395-410.

55) Ryan P, Maher K, Murphy B, Hurley GD, Fitzpatrick JM. Experimental partial ureteric obstruction: pathophysiological changes in upper tract pressure and renal blood flow. J Urol 1987;138: 674–678.

56) Murphy G, Scott W. The renal hemodynamic response to acute and chronic ureteral obstruction. J Urol 1966;95:636–657.

57) Moody T, Vaughan E, Gillenwater J. Relationship between renal blood flow and ureteral pressure during 18 hours of total unilateral ureteral occlusions. Invest Urol 1975;13:246–251.

58) Badr K, Brenner B. Renal circulatory and nephron function in experimental obstruction of the urinary tract. In: Brenner BM, Lazarus J, eds. Acute renal failure. New York: Churchill Livingstone, 1988:91–118.

59) Klahr S, Buerkert J, Morrison A. Urinary tract obstruction. In: Brenner BM, Rector F, eds. The kidney. Philadelphia: Saunders, 1986:156–168.

60) Klahr S. Pathophysiology of obstructive nephropathy: a 1991 update. Semin Nephrol 1991;11: 156–168.

61) Yarger W, Griffith L. Intrarenal hemodynamics following chronic unilateral ureteral obstruction in the dog. Am J Physiol 1974;65:816–826.

62) Ichikawa I, Brenner B. Local intrarenal vasoconstrictor– vasodilator interactions in mild partial ureteral obstruction. Am J Physiol 1979;236: F131–F140.

63) Fitzgerald G, Murray R, Price P, Catella F. The molecular, biochemical and human pharmacology of thromboxane A2 in renal disease. Adv Exp Med Biol 1989;259:325–360.

64) Wilson D. Pathophysiology of obstructive nephropathy. Kidney Int 1980;18:325–360.

65) Albrightson C, Evers A, Griffin A, Needleman P. Effect of endogenously produced leukotrienes and thromboxane on renal vascular resistance in rabbit hydronephrosis. Circ Res 1987;61:514–522.

66) Kawasaki A, Needleman P. Contribution of thromboxane to renal resistance changes in the isolated perfused hydronephrotic rabbit kidney. Circ Res 1982;50:486–490.

67) Rowin BH, Harris KP, Morrison A, Klahr S, Schreiner GF. Renal cortical release of a specific macrophage chemoattractant in response to ureteral obstruction. Lab Invest 1990;63:213–220.

68) Yarger W, Schocken D, Harris R. Obstructive nephropathy in the rat. J Clin Invest 1980; 227:400–412.

69) Curry T.S, Dowdey J.E, Murry R.C. Ultrasound. In: Curry T.S , Dowdey J.E, Murry R.C, editors. Christenson's Physics of Diagnostic Radiology. 4th ed. Philadelphia: Lea and Febiger; 1990.p. 323-371.

70) Meire H., Farrant P. A historical overview. In: Meire H., Farrant P. editors. Basic Ultrasound: A historical overview. John Wiley and Sons Publications; 1995.p.1-7

71) Donald I., Brown T.G. Demonstration of tissue interfaces within the body by ultrasonic echosounding. Br J Radiol 1961 Sep;34:539–546.

72) Howry D.H brief atlas of ultrasonic radiologic results. Radiologic Clinics of North America 1965Dec; 3(3):433-452.

73) Bo W.J,Krueger W.A.Anatomic concepts of the urogenital system:Gross and cross sectional anatomy. In: Resnick M.I, Sanders R.C. editors. Ultrasound in Urology. Baltimore: Williams and Wilkins; 1979.p. 37.

74) Brandt TD, Neiman H , Dragowski M J, Bulawa W, Claycamp G. Ultrasound assessment of normal renal dimensions. Ultrasound Med 1982 Mar; 1(2): 49- 52.

75) Cronan. J. J and A. T. Rosenfield. Normal size of the adult kidney by real time ultrasound. Am J Roentgenol 1990; 154 : 195.

76) Ninan V. T., Koshi K. T., Niyamthullah M.M, Jacob CK, Gopalakrishnan G, Pandey AP et al. A comparative study of methods of estimating renal size in normal adults. Nephrol Dial Transplant 1990; 5(10) : 851 - 854.

77) Allan. P. L .Medical diseases of the kidney. In: Cosgroove. D, Meire H and Dewbury K,editors. Clinical ultrasound a comprehensive text: Abdominal and General Ultrasound Volume II. Edinburgh : Churchill Livingstone; 1993.p. 513 - 524.

78) Rosenfield A.T, Taylor K.J.W., Crade M, DeGraaf CS. Anatomy and pathology of the Kidney by gray scale ultrasound. Radiology 1978 Sept; 128(3):737-744.

79) Huntington. D. K., Hill S. C., Hill M. C. Sonographic manifestations of medical renal disease. Semin in Ultrasound CT MR 1991; 12 (4) : 290 – 307.

80) Cook. J. H, Rosenfield A. T. and Taylor K. J. Ultrasonic demonstration of intrarenal anatomy. AJR Am-J- Roentegenol 1977Nov; 129(5): 831 - 835.

81) Hricak. H. Renal Medical Disorders : The role of sonography. In:Sanders R C, editor. Ultrasound Annual. New York: Lippincott-Raven Publishers; 1982.p.43.

82) Cunningham J.J. Nonobstuctive fragmentation of renal central pelvicalyceal echo complex.Urology 1979Jan; 13(1):99-102.

83) Saunders R. C., Sandra. L. Hundley. Renal failure, In: Saunders. R.C, editor, Clinical Sonography. A Practical guide. Boston: Little, Brown & Company; 1984.p.299-310.

84) Emamian S.A, Nielsen M.B, Pederson J.F, Ytte L. Kidney Dimensions at Sonography: Correlation with Age, Sex and Habitus in 665 Adult Volunteers. AJR Am J Roentgenol 1993 Jan; 160(1):83-86.

85) Hricak H, Slovis T, Callen C, Romanski R. Neonatal kidneys. Sonographic anatomic correlation. Radiology 1983 Jun; 147(3):699-702.

86) Lafortune. M., Constantin A., Breton G. Vallee C. Sonography of the hypertrophied column of Bertin. AJR Am J Roentgenol 1986 Jan; 146(1): 53 - 56.

87) Sanders R.C, Jeck D.C. B Scan in the evaluation of renal failure. Radiology 1976 Apr; 119(1):199-202.

88) Platt J, Ellis J, Rubin J. Examination of native kidneys with duplex Doppler ultrasound. Semin Ultrasound CT MR 1991;12:308–318.

89) Keogan M, Kliewer M, Hertzberg B, DeLong DM, Tupler RH, Carroll BA. Renal resistive indexes: variability in Doppler US measurement in a healthy population. Radiology 1996;199:165–169.

90) Norris C, Pfeiffer J, Rittgers S, Barnes RW. Noninvasive evaluation of renal artery stenosis and renovascular resistance: experimental and clinical studies. J Vasc Surg 1984;1:192–201.

91) Gottlieb R, Luhmann K, Oates R. Duplex ultrasound evaluation of normal native kidneys with urinary tract obstruction. J Ultrasound Med 1989; 8:609–611.

92) Ryan P, Maher K, Murphy B, Hurley GD, Fitzpatrick JM. Experimental partial ureteric obstruction: pathophysiological changes in upper tract pressure and renal blood flow. J Urol 1987;138: 674–678.

93) Murphy G, Scott W. The renal hemodynamic response to acute and chronic ureteral obstruction. J Urol 1966;95:636–657.

94) Moody T, Vaughan E, Gillenwater J. Relationship between renal blood flow and ureteral pressure during 18 hours of total unilateral ureteral occlusions. Invest Urol 1975;13: 246–251.

95) Badr K, Brenner B. Renal circulatory and nephron function in experimental obstruction of the urinary tract. In: Brenner BM, Lazarus J, eds. Acute renal failure. New York: Churchill Livingstone, 1988:91–118.

96) Klahr S, Buerkert J, Morrison A. Urinary tract obstruction. In: Brenner BM, Rector F, eds. The kidney. Philadelphia: Saunders, 1986:156–168.

97) Paul Allan, Paul A. Dubins, W. Norman McDicken, Myron A. Pozniak. Meghan MAteer, editor. Clinical Doppler Ultrasound. 2nd edition. Churchill Livingstone company; 2006.p.208-209.

98) Ellenbogen PH, Scheible FW, Talner LB, Leopold GR. Sensitivity of gray-scale ultrasound in detecting urinary tract obstruction. AJR Am J Roentgenol 1978;130:731-733.

99) Laing FC, Jeffrey RB Jr, Wing VW. USG versus excretory urogra- phy in evaluating acute flank pain. Radiology 1985;154:613-6.

100) Mostbeck GH, Zontsich T, Turetschek K. Ultrasound of the kidney: Obstruction and medical diseases. Eur Radiol 2001;11:1878-89.

CONSENT FORM

பூ. சா. கோ மருத்துவக் கல்லூரி மற்றும் ஆராய்ச்சி நிறுவனம், கோவை

மனித நெறிமுறைக் குழு

ஒப்புதல் படிவம்

தேதி:

மரு. ஷம்ஸா காப்புமுகத் முகமது, ஆகிய நான் பூ. சா. கோ மருத்துவக் கல்லூரியின் / மருத்துவமனையின் மருந்தியல் துறையின் கீழ், **"மீயொலி வண்ணப் பரிசோதனை (டாப்ளர்) மூலம் அடைப்புள்ள மற்றும் அடைப்பு இல்லாத சிறுநீரகத்தின் மதிப்பீடுகளை ஒப்பிடுவது"** என்ற தலைப்பில் ஆய்வு மேற்கொள்ள உள்ளேன்.

என் ஆய்வு வழிகாட்டி: மரு. இளங்கோ .N DMRD, MDRD.,

ஆய்வு மேற்கொள்வதற்கான அடிப்படை:

MD மருத்துவ மேற்படிப்பு பாடத்திட்டத்தின்படி இரண்டு ஆண்டுகளுக்குள் ஆராய்ச்சி செய்யப்பட வேண்டும். மீயொலி வண்ணப் பரிசோதனை (டாப்ளர்) மூலம் அடைப்புள்ள மற்றும் அடைப்பு இல்லாத சிறுநீரகத்தின் மதிப்பீடுகளை ஒப்பிடுவதே என் ஆராய்ச்சியாகும்.

மீயொலி வண்ணப் பரிசோதனை (டாப்ளர்) மூலம் எதிர்ப்புதிறன் அளவீடுகளைக் கொண்டு ஆர். ஐ சிறுநீரக அடைப்புகளை அறுவை சிகிச்சையின்றி கண்டறிய முடியும்.

இப்பரிசோதனை மூலம் சிறுநீரக அடைப்பால் வரும் வயிற்று வலியையும் மற்ற காரணங்களால் வரும் வயிற்று வலிகளையும் கண்டறிய முடியும். இதன் மூலம் தேவையில்லாமல் சிறுநீரகக் கல்லுக்காக சி.டி ஊடுகதிர் பரிசோதனை செய்வதைக் குறைக்க முடியும்.

மேலும் இந்த அளவீடுகள் நோய்களை கண்டறியும் மற்றும் சிகிச்சை திட்டமிடவும் உதவுகின்றன.

ஆய்வின் நோக்கம்:

முதன்மை நோக்கம்:

- மீயொலி வண்ணப் பரிசோதனை (டாப்ளர்) மூலம் அடைப்புள்ள மற்றும் அடைப்பு இல்லாத சிறுநீரகத்தின் மடிப்பீடுகளை ஒப்பிடுதல்.
- சிறுநீரக அடைப்புகளை கண்டறிவதில் டாப்ளர் அளவீடுகளின் பங்கு மற்றும் பயன்பாடு குறித்து ஆராய்தல்.

இரண்டவது நோக்கம்:

அடைப்பின் தன்மை இடம் மற்றும் நேர இடைவெளி ஆகியவை மீயொலி வண்ணப் பரிசோதனை (டாப்ளர்) மதிப்பீடுகள் மீது ஏற்படுத்தும் தாக்கம் குறித்து ஆராய்தல்.

ஆய்வில் பங்கு பெறும் நபர்களின் எண்ணிக்கை: 40

ஆய்வில் பங்கு பெறுவோர் மற்றும் வயது: அனைத்து வயதினரும்

ஆய்வு மேற்கொள்ளும் இடம்: பூ. சா. கோ மருத்துவ கல்லூரி மருத்துவமனை, கோயமுத்தூர்

இந்த ஆய்வில் எங்களுடன் ஒத்துழைக்குமாறு கேட்டுக்கொள்கிறோம். நாங்கள் சில தகவல்களை இந்த ஆய்விற்காக சேகரிக்க உள்ளோம்.

ஆய்வு செய்யப்படும் முறை:

முதன்மை நேர்காணல்: _____

இந்த ஆய்வில் கிடைக்கும் தகவல்கள் **3 வருடங்கள்** பாதுகாக்கப்படும். இந்த தகவல்கள் வேறு ஆய்விற்குப் பயன்படுத்தப் படும்/பயன்படுத்தப் பட மாட்டாது.

மருத்துவ பரிசோதனைகள்:

இரத்த மாதிரி சேகரிப்பு:

இரத்த மாதிரி எடுப்பது வழக்கமான சிகிச்சைக்காகவோ அல்லது இந்த ஆய்விற்காகவோ:

ஆய்வினால் ஏற்படக் கூடிய அசௌகரியங்கள் / பக்க விளைவுகள்: இதனால் எந்த அசௌகரியமோ, பக்க விளைவுகளோ ஏற்படாது.

இரத்த மாதிரிகள் ஆய்விற்குப் பின் பாதுகாத்து வைக்கப்படுமா? ஆம் / இல்லை, அழிக்கப்படும்: **இல்லை**

சேகரிக்கப்பட்ட இரத்தம் விற்கப்படுமா? ஆம் / இல்லை **இல்லை**

சேகரிக்கப்பட்ட இரத்தம் வேறு நிறுவனத்துடன் பகிர்ந்து கொள்ளப்படுமா? ஆம் / இல்லை: **இல்லை**

மருந்துகள் ஏதேனும் கொடுக்கப் பட்டிருந்தால் அவை பற்றிய விவரம் (கொடுக்கப்பட்ட காரணம், காலம், பக்க விளைவுகள், பயன்கள்) **இல்லை**

மருந்துகள் கொடுக்கப்படுவது வழக்கமான சிகிச்சை முறையா? ஆம் / இல்லை (ஆம் என்றால் இந்த குறிப்பிட்ட மருந்து கொடுக்கப்பட்டதன் கரணம்)

ஆய்வில் பங்குபெறுவதால் ஏற்படும் பலன்கள்:
அடைபட்ட சிறுநீரகத்தின் வண்ணப் பரிசோதனை அளவீடுகள் (ஆர்.ஐ) நோய்க்காரணிகளை கண்டறிவதிலும் சிகிச்சைமுறை திட்டமிடுவதிலும் துணை புரிகின்றன.

ஆய்வின் முடிவுகள் எந்த முறையில் பயன்படுத்தப்படும்?
ஆய்வின் முடிவுகள், அடுத்தகட்ட ஆராய்ச்சிகளுக்கும், மருத்துவ ஆய்வு பத்திரிக்கைகளில் வெளியிடுவதற்கும் பயன்படுத்துப்படும்.

இந்த ஆய்வின் கேள்விகளுக்கு பதிலளிப்பதோ, இரத்த மாதிரிகள் அல்லது திசு மாதிரிகள் எடுப்பதிலோ உங்களுக்கு ஏதேனும் அசௌகரியங்கள் இருந்தால், எந்த நேரத்தில் வேண்டுமானாலும் ஆய்விலிருந்து விலகிக்கொள்ளும் உரிமை உங்களுக்கு உண்டு. எப்பொழுது வேண்டுமானாலும் ஆய்விலிருந்து விலகும் உரிமை உங்களுக்கு உள்ளது. ஆய்விலிருந்து விலகிக்கொள்வதால் உங்களுக்கு அளிக்கப்படும் சிகிச்சை முறையில் எந்த வித பாதிப்பும் இருக்காது என்று உங்களுக்கு உறுதியளிக்கிறோம். மருத்துவ மனையில் நோயாளிகளுக்கு அளிக்கப்படும் சேவைகளை நீங்கள் தொடர்ந்து பெறலாம். இந்த ஆய்வில் பங்கேற்க ஒப்புக்கொள்ளுவதால் வேறு எந்த விதமான கூடுதலான பலனும் உங்களுக்குக் கிடைக்காது. நீங்கள் அளிக்கும் தகவல்கள் இரகசியமாக வைக்கப்படும். ஆய்வில் பங்கேற்பவர்கள் பற்றியோ அவர்கள் குடும்பத்தைப் பற்றியோ எந்தத் தகவலும் எக்காரணம் கொண்டும் வெளியிடப்படாது என்று உறுதியளிக்கிறோம். நீங்கள் அளிக்கும் தகவல்கள் / இரத்த மாதிரிகள் / திசு மாதிரிகள் அங்கீகரிக்கப்பட்ட ஆய்விற்கு மட்டுமே பயன்படுத்தப்படும். இந்த ஆய்வு நடைபெறும் காலத்தில் குறிப்பிடத்தகுந்த புதிய கண்டுபிடிப்புகள் அல்லது பக்க விளைவுகள் ஏதும் ஏற்பட்டால் உங்களுக்குத் தெரிவிக்கப்படும். இதனால் ஆய்வில் தொடர்ந்து பங்கு பெறுவது பற்றிய உங்கள் நிலைப்பாட்டை நீங்கள் தெரிவிக்க ஏதுவாகும்.

ஆய்வுக்குட்படுபவரின் ஒப்புதல்: இந்த ஆய்வைப் பற்றிய மேற்கூறிய தகவல்களை நான் படித்து அறிந்து கொண்டேன் / ஆய்வாளர் படிக்கக் கேட்டுத் தெரிந்து கொண்டேன். ஆய்வினைப் பற்றி நன்றாகப் புரிந்து கொண்டு இந்த ஆய்வில் பங்கு பெற ஒப்புக்கொள்கிறேன். இந்த ஆய்வில் பங்கேற்பதற்கான எனது ஒப்புதலை கீழே கையொப்பமிட்டு . கை ரேகை பதித்து நான் தெரிவித்துக் கொள்கிறேன்.

பங்கேற்பாளரின் பெயர், முகவரி:

பங்கேற்பாளரின் கையொப்பம் / கை ரேகை / சட்டப்பூர்வ பிரதிநிதியின் கையொப்பம்:

தேதி :

ஆய்வாளரின் கையொப்பம் :
தேதி :

ஆய்வாளரின் தொலைபேசி எண்: 9791890685
மனித நெறிமுறைக் குழு அலுவலகத்தின் தொலைபேசி எண்:
அலுவலக நேரத்தில்0422 2570170 Extn.: 5808
அலுவலக் நேரத்திற்குப்பின்: 9865943043

MASTER CHART

SL NO	NAME	OP NO	AGE	SEX	RIGHT KIDNEY RI VALUE	LEFT KIDNEY RI VALUE	DELTA RI	OBSTRUCTION		LEVEL OF OBSTRUCTION	GREY SCALE FINDING IN OBSTRUCTED KIDNEY	CAUSE OF OBSTRUCTION	RENAL PARENCHYMAL THICKNESS					
					MEAN	MEAN		RIGHT KIDNEY	LEFT KIDNEY			USG/ CT	RIGHT KIDNEY			LEFT KIDNEY		
													UP	MP	LP	UP	MP	LP
1	Palanisamy	O16017209	55	M	0.57	0.695	0.12		+	Left distal ureteric calculus	No hydronephrosis	CT KUB - 5mm calculus	17mm	20mm	18mm	16.6 mm	30mm	18.7mm
2	Parveen Begum	O16017267	54	F	0.51	0.73	0.22		+	Left distal ureteric calculus	Mild hydronephrosis	CT KUB - 7 mm calculus	15.6mm	23mm	19.8mm	15.8mm	21.2mm	17.8mm
3	Renuka	O15061805	22	F	0.75	0.63	0.12	+		Right proximal ureteric calculus	Mild hydronephrosis	USG - 5mm calculus	20mm	20mm	18mm	17mm	18mm	16mm
4	Abdul Majeed	O15010970	79	M	0.58	0.72	0.13		+	Left distal ureteric calculus	Mild hydronephrosis	USG - 5mm calculus	18mm	16mm	18mm	18mm	16mm	15mm
5	Ramesh kumar	O16037166	36	M	0.52	0.72	0.2		+	Left distal ureteric calculus	Mild hydronephrosis	USG - 6mm calculus	20mm	20mm	17mm	13mm	23mm	17mm
6	Pappathy	O08085674	52	F	0.76	0.6	0.15	+		Right distal ureteric calculus	Mild hydronephrosis	CT - 3mm calculus	20mm	16mm	15mm	15mm	24mm	15.3mm
7	Kumar	O16046379	39	M	0.7	0.52	0.17	+		Right distal ureteric calculus	No hydronephrosis	USG 4 mm calculus	19.2mm	20mm	17mm	12mm	19mm	15mm
8	Duraisamy	O12040164	62	M	0.53	0.72	0.19		+	Left proximal ureteric calculus	Mild hydronephrosis	USG - 9 mm calculus	18mm	17.5mm	18mm	15mm	17mm	18mm
9	Kathiresan	O16055488	19	M	0.82	0.47	0.35	+		Right distal ureteric calculus	No hydronephrosis	CT - 3mm calculus	21mm	20mm	20mm	17mm	21mm	20mm
10	Senthil kumar	O16061101	46	M	0.48	0.84	0.36		+	Left proximal ureteric calculus	Mild hydronephrosis	CT - 9 mm calculus	18mm	26mm	18mm	15mm	18mm	23mm
11	Thangamani	O16061656	58	F	0.56	0.76	0.2		+	Left proximal ureteric calculus	Mild hydronephrosis	CT -6mm calculus	12mm	17mm	18mm	11mm	22mm	15mm
12	Mithresh kannan	O12048819	23	M	0.47	0.71	0.24		+	Left distal ureteric calculus	Mild hydronephrosis	CT - 5 mm calculus	21mm	20mm	19mm	19.5mm	21mm	19.6mm
13	Sakunthala	O16002676	61	F	0.57	0.76	0.19		+	Left proximal ureteric calculus	Mild hydronephrosis	CT - 9 mm calculus	18.6mm	16mm	15mm	17mm	16mm	18mm
14	Janaki R	O16075791	57	F	0.48	0.84	0.36		+	Left proximal ureteric calculus	Mild hydronephrosis	CT - 6 mm calculus	12.9mm	23.1mm	16.6mm	17mm	26mm	18mm
15	Karthikeyan	O16082658	31	M	0.71	0.46	0.25	+		Right distal ureteric calculus	Mild hydronephrosis	CT - 5 mm calculus	19mm	16mm	15mm	18mm	17mm	15mm
16	Rukkmani	O16085034	44	F	0.48	0.7	0.22		+	Left distal ureteric calculus	Mild hydronephrosis	CT - 5 mm calculus	13mm	18mm	14mm	15mm	14mm	16mm
17	Palanisamy N	O16086961	70	M	0.64	0.73	0.09		+	Left proximal ureteric calculus	Mild hydronephrosis	CT - 8 mm calculus	21mm	17mm	20mm	16.7mm	16.4mm	20mm
18	Krishnaswamy	O16089443	49	M	0.54	0.75	0.21		+	Left distal ureteric calculus	Mild hydronephrosis	USG - 7 mm calculus	14.9mm	15.4mm	15.8mm	16.4mm	18.2mm	16.2mm
19	Rajendran	O17003015	44	M	0.74	0.58	0.16	+		Right distal ureteric calculus	Mild hydronephrosis	CT- 6mm calculus	21mm	22mm	20mm	21mm	18mm	20mm
20	Angamuthu	O12054937	65	M	0.75	0.61	0.14	+		Right proximal ureteric calculus	Mild hydronephrosis	CT -6 mm calculus	20mm	24mm	15mm	19.3mm	19.5mm	16.4mm

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					MEAN	MEAN		RIGHT KIDNEY	LEFT KIDNEY			USG/ CT	RIGHT KIDNEY			LEFT KIDNEY		
													UP	MP	LP	UP	MP	LP
21	Devaraj	O17012679	53	M	0.79	0.56	0.23	+		Right distal ureteric calculus	Mild hydronephrosis	CT 5 mm calculus	21 mm	29 mm	26 mm	22 mm	27 mm	19 mm
22	Ranganathan	O17004892	46	M	0.52	0.81	0.29		+	Left proximal ureteric calculus	Moderate hydronephrosis	CT - 10 mm calculus	16mm	24 mm	14 mm	20 mm	19 mm	18 mm
23	Sivakala	O17018425	53	F	0.6	0.74	0.14		+	Left distal ureteric calculus	Mild hydronephrosis	USG -5 mm calculus	17mm	18mm	16mm	19 mm	15 mm	18mm
24	Murukesh	O17038009	47	M	0.56	0.81	0.25		+	Left proximal ureteric calculus	Mild hydronephrosis	CT -6 mm calculus	22mm	18mm	17mm	18mm	19mm	21mm
25	Sengodu	O12078490	67	M	0.8	0.51	0.29	+		Right proximal ureteric calculus	Mild hydronephrosis	CT - 15mm calculus	16mm	20mm	15mm	18mm	18mm	16mm
26	Petchiammal	O17038042	50	F	0.79	0.56	0.23	+		Right proximal ureteric calculus	Moderate hydronephrosis	USG - 15 mmcalculus	19mm	19mm	15mm	16mm	18mm	15mm
27	Santhakumari	O17028474	45	F	0.81	0.56	0.25	+		Right proximal ureteric calculus	Mild hydronephrosis	CT - 13 mm calculus	19mm	15mm	17mm	18mm	18mm	19mm
28	Balraj R	O17028259	49	M	0.86	0.47	0.39	+		Right proximal ureteric calculus	Mild hydronephrosis	CT- 5 mm calculus	17mm	15mm	19mm	18mm	16mm	18mm
29	Ramadass V	O17028660	52	M	0.54	0.83	0.29		+	Left distal ureteric calculus	Mild hydronephrosis	CT - 12 mm calculus	18mm	17mm	17mm	18mm	19mm	18mm
30	Nagarajn	O16087180	53	M	0.73	0.61	0.11	+		Right distal ureteric calculus	Mild hydronephrosis	CT - 5mm calculus	18mm	15mm	15mm	18mm	17mm	16mm
31	Janakiraman B.N	O17029650	52	M	0.57	0.84	0.27		+	Left distal ureteric calculus	Mild hydronephrosis	CT - 8mmcalculus	15mm	16mm	17mm	20mm	17mm	16mm
32	Siddharth G	O17030606	20	M	0.53	0.83	0.3		+	Left distal ureteric calculus	Mild hydronephrosis	CT 5 mm calculus	17mm	19mm	17mm	21mm	19mm	21mm
33	Franais Xavier	O17033713	25	M	0.8	0.51	0.29	+		Right proximal ureteric calculus	Mild hydronephrosis	CT 8mm calculus	19mm	17mm	17mm	20mm	17mm	17mm
34	Vallinayagam	O15049215	49	F	0.86	0.55	0.31	+		Right proximal ureteric calculus	Mild hydronephrosis	CT 10mm calculus	18mm	16mm	16mm	17mm	15mm	15mm
35	Shalini	O17033014	17	F	0.56	0.8	0.24		+	Left distal ureteric calculus	No hydronephrosis	CT - 10mm calculus	16mm	15mm	16mm	15mm	18mm	16mm
36	Murugavel	O16082758	68	M	0.86	0.52	0.35	+		Right distal ureteric calculus	Mild hydronephrosis	CT - 5mm calculus	21mm	20mm	21mm	16mm	20mm	15mm
37	Ganeshan M	O17003795	51	M	0.52	0.77	0.25		+	Left proximal ureteric calculus	Moderate hydronephrosis	CT -14 mm calculus	17mm	19mm	18mm	17mm	19mm	17mm
38	Shanmugavalli	O13008393	42	F	0.72	0.59	0.15		+	Left proximal ureteric calculus	Moderate hydronephrosis	CT -15 mm calculus	20mm	16mm	18mm	22mm	20mm	22mm
39	Yazhini	O17026027	18	F	0.8	0.52	0.27	+		Right proximal ureteric calculus	Mild hydronephrosis	CT - 5 mm calculus	15mm	16mm	19mm	15mm	16mm	18mm
40	Subramanian	O17018116	60	M	0.49	0.85	0.36		+	Left proximal ureteric calculus	Mild hydronephrosis	CT - 6mm calculus	16mm	15mm	19mm	15mm	18mm	17mm

SL NO	NAME	OP NO	AGE	SEX	RIGHT KIDNEY RI VALUE	LEFT KIDNEY RI VALUE	DELTA RI	OBSTRUCTION		LEVEL OF OBSTRUCTION	GREY SCALE FINDING IN OBSTRUCTED KIDNEY	CAUSE OF OBSTRUCTION	RENAL PARENCHYMAL THICKNESS					
					MEAN	MEAN		RIGHT KIDNEY	LEFT KIDNEY			USG/ CT	RIGHT KIDNEY			LEFT KIDNEY		
													UP	MP	LP	UP	MP	LP
41	Murugeshan	O17057020	34	M	0.76	0.5	0.26	+		Right proximal ureteric calculus	Mild hydronephrosis	CT - 8mm calculus	21mm	20mm	21mm	16mm	20mm	15mm
42	Vishnu	O14080967	20	M	0.72	0.53	0.19	+		Right distal ureteric calculus	Mild hydronephrosis	USG - 5 mm calculus	15mm	16mm	16mm	15mm	16mm	17mm
43	Raja V	O17029676	31	M	0.79	0.52	0.27	+		Right proximal ureter calculs	Mild hydronephrosis	CT - 6 mm calculus	20mm	18mm	21mm	17mm	15mm	16mm
44	Mahadeswara Prabhu	O11082377	41	M	0.46	0.84	0.38		+	Left proximal ureteric calculus	Mild hydronephrosis	CT 6mm calculus	17mm	17mm	18mm	18mm	15mm	17mm
45	Kumarasamy R	O17043810	66	M	0.83	0.45	0.38	+		Right proximal ureteric calculus	Mild hydronephrosis	CT - 5mm calculus	15mm	17mm	16mm	15mm	16mm	15mm
46	Dhanapal	O15049313	21	M	0.48	0.75	0.27		+	Left distal ureteric calculus	Mild hydronephrosis	CT 5 mm calculus	19mm	18mm	18mm	20mm	17mm	15mm
47	Jothi R	O17003339	46	F	0.82	0.48	0.34	+		Right distal ureteric calculus	Mild hydronephrosis	CT - 6mm calculus	20mm	16mm	16mm	20mm	17mm	15mm
48	Meena R	O17026926	42	F	0.79	0.53	0.26	+		Right proximal ureteric calculus	Mild hydronephrosis	CT - 6mm calculus	16mm	16mm	18mm	17mm	14mm	13mm
49	Prabhakaran	O17038160	25	F	0.5	0.81	0.31		+	Left distal ureteric calculus	Moderate hydronephrosis	CT - 7mm calculus	16mm	15mm	17mm	15mm	16mm	18mm
50	Kannan M	O17004810	48	M	0.78	0.56	0.22	+		Right distal ureter calculus	Mild hydronephrosis	CT - 5mm calculus	16mm	17mm	16mm	16mm	17mm	16mm
51	Sekar	O17004365	37	M	0.48	0.81	0.33		+	Left distal ureteric calculus	Mild hydronephrosis	CT - 9 mm calculs	17mm	18mm	13mm	15mm	20mm	20mm
52	Karthiga	O17007424	19	F	0.48	0.78	0.3		+	Left proximal ureteric calculus	Mild hydronephrosis	CT- 6 mm calculus	17mm	17mm	16mm	18mm	15mm	16mm
53	Sivapriyanka	O17066897	21	F	0.55	0.8	0.26		+	Left distal ureteric calculus	Mild hydronephrosis	CT - 7mm calculus	19mm	18mm	17mm	18mm	17mm	16mm
54	Kowsalya	O17065803	19	F	0.71	0.53	0.18	+		Right distal ureteric calculus	Mild hydronephrosis	USG - 5 mm calculus	17mm	15mm	16mm	19mm	18mm	16mm